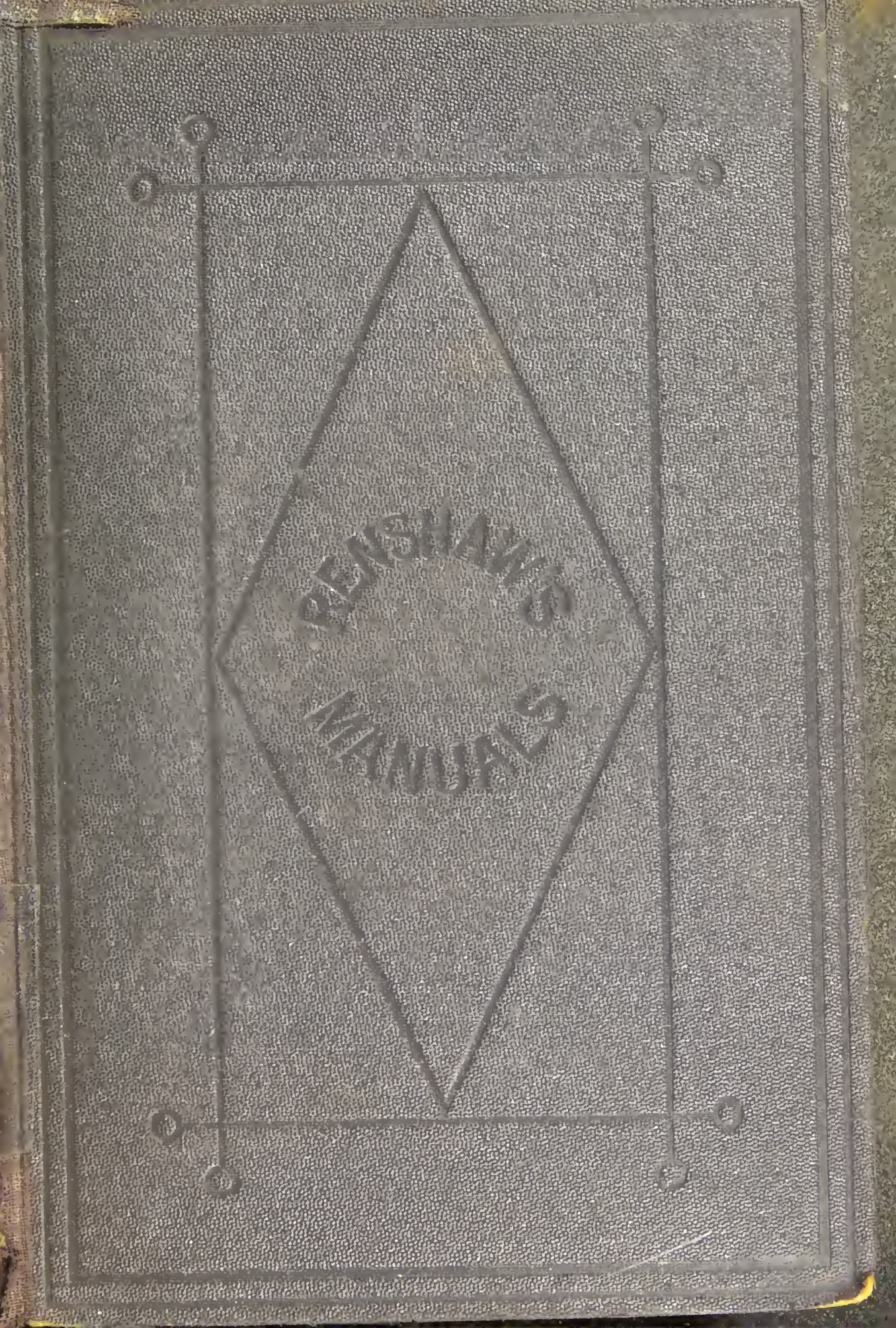

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THE PRACTICE AND THEORY OF MEDICINE

VOL. I.

UNIVERSITY OF LEEDS

A MANUAL OF THE PRACTICE AND THEORY OF MEDICINE

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ETC., ETC.

IN TWO VOLUMES

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TO

SIR CLIFFORD ALLBUTT, K.C.B.

M.D., LL.D., F.R.C.P., D.SC., F.R.S., F.S.A., F.L.S.

Regius Professor of Physic, University of Cambridge

IN ADMIRATION OF HIS GENIUS AS AN
ORIGINAL OBSERVER AND PROFOUND THINKER
WHOSE PHILOSOPHIC AND CLASSIC CONTRIBUTIONS TO
ALMOST EVERY DEPARTMENT OF MEDICINE
HAVE STIMULATED RESEARCH AND LEFT A
LASTING IMPRESSION UPON

The Science of the Healing Art

THESE VOLUMES ARE GRATEFULLY DEDICATED
BY THE AUTHOR

SCHOOL OF MEDICINE,
UNIVERSITY OF LEEDS.

PREFACE

At the earnest solicitations of many medical friends who felt that they had been assisted in their daily routine by the 'Dictionary of Treatment,' I have attempted to embody in the present volumes the experience of a quarter of a century of clinical observation and medical teaching in the wards of the Royal Victoria Hospital.

The aim of the work is to afford to the busy practitioner in the most convenient and condensed form a series of independent articles upon the different diseases (including those of the skin) which fall under the province of the physician. 'A Dictionary of Medicine' or 'A Medical Index' would obviously have been a more correct and suitable title for the work; but fearing that such a nomenclature would lead to confusion as regards the 'Dictionary of Treatment,' to which the present work may be accepted as an introduction, it was found necessary to adopt the more pretentious title of 'The Practice and Theory of Medicine.' With the view of making the work as helpful as possible to the busy practitioner, it has been divided into two volumes, each containing a complete Index to both, the whole being condensed into such dimensions as in an emergency may find a place in the instrument-bag or travelling equipment of the physician.

The dictionary-form of the work necessitates a certain amount of repetition, as the aim of each article is to give a condensed and more or less isolated description of the etiology, pathology, symptomatology, diagnosis, and a brief *résumé* of the treatment of each medical affection. That portion of each article which deals with the subject of differential diagnosis is planned so that, as far as possible, cross-references are avoided, the reader being enabled to contrast the more

important distinguishing clinical features of diseases liable to be confused with each other without having to turn to different portions of the work. The selection of the diseases to be differentiated in each case may often seem an arbitrary one to the reader, but it has been dictated by the experience of many years of clinical teaching and of cross-examination of the senior student by the bedside, as well as by the experience derived from intercourse with the younger members of the profession in the routine of consultation-practice. Whilst mainly relying upon personal observation and practical experience for the description of the affections daily coming under the eye of the physician and clinical teacher, the more recent special knowledge accumulated by original investigators in the different departments of clinical medicine has been as fully utilised as the limited space permits, and, except by inadvertence, due acknowledgment of indebtedness to authority will be found recorded in each instance.

Statistics are but lightly availed of throughout the work—a fact which may strike the reader as a regrettable omission ; but the materials for the compilation of *post-mortem* and other records, to be of real value in the advancement of medical science, can only be accumulated by expert assistance and methods which were not in every case at my disposal. Statistics, to be a help and not a cause of bewilderment to the practitioner, must be compiled from a survey of wide extent and from records whose accuracy should defy challenge. In the condensed survey of the theory and practice of medicine attempted in the present work their use is confined to the elucidation of general principles.

My thanks and indebtedness are sincerely acknowledged to Dr. Victor Fielden and to Dr. Thomas Houston for their invaluable assistance in forwarding the volumes through the press—especially to the former for the exact and replete Index and to the latter for his kindness in drawing my attention to several recent researches in the domain of bacteriology which might otherwise have escaped notice.

WILLIAM WHITLA.

LENNOXVALE, BELFAST :
April 1908.

ACNE

This is an affection resulting from an inflammatory condition in and around the sebaceous glands which exist in connection with the rudimentary hairs on the nose, cheeks, forehead, external ears, nucha, shoulders, and sternal region.

Etiology and Pathology.—The disease usually first shows itself about the period of puberty, the majority of cases occurring between the ages of fourteen and twenty years, when the stimulus to new hair-growth is most active in association with the development of the reproductive organs.

Males are as a rule more frequently and more severely affected than females. The disease shows a tendency to disappear after the full development of the hairs of the beard and body, but it may remain long afterwards. It occurs often in young male adults who are in the most typical robust health. Vaso-motor disturbances are obviously the exciting or predisposing cause in some cases, the condition being frequently associated with the tendency to blushing. Dietetic errors probably only play a *rôle* in the affection after it has become established; in some individuals these apparently aggravate it, and seem to determine the process of suppuration. A similar result may often be seen to follow irregularities in menstruation.

A coarse texture of skin is found in many patients. The question of a microbic origin of the disease has been often debated, and Jackson maintains that it will be proven that the condition is the result of an infection by a special form of micro-organism operable between the ages of twelve and twenty-four, and Unna holds that all the stages of acne are due to a specific bacillus. The difficulty in deciding the correctness of the microbic theory arises from the fact that numerous organisms—staphylococci, bacilli, &c.—are found in the comedones, resulting from plugging of the sebaceous ducts; these by many authorities are still regarded as accidental or secondary, and not as the primary cause. The frequent association of the condition with seborrhœa of the scalp is held by some to be a proof of the microbic theory. The view that the suppurative process is originated in the preceding comedones is gaining ground, and has led to advances in the treatment of the disease, to be referred to later on. The parasite *Demodex folliculorum* is found occasionally in the sebaceous duct; it is, however, accepted as playing no part either in the production of comedones or of acne.

Symptoms.—Various varieties of acne are recognised. The typical form of the disease is known as *acne vulgaris*. This arises usually as a sequel to the condition called *acne punctata* or comedone formation, which is not an inflammatory affection. The comedone is a small mass or plug of sebaceous material occupying the duct of a sebaceous gland in connection with a rudimentary hair in the usual acne region. Payne points out that the accumulation of sebum in the duct is not due to obstruction, since the orifice is patent ; the increased consistency of the sebaceous secretion is probably the main factor. The plug at its superficial extremity is usually of a dark colour, due to the presence of dirt or to cornification of the cells of the epidermis or to pigmentation of an obscure origin ; hence its usual popular designation of ' black-head.' By squeezing the skin on each side of the comedone the contents are pressed out in the shape of a small grub, in which the demodex can sometimes be recognised, and numerous cutaneous micro-organisms are commonly also present. In both comedone formation and true acne there is a marked greasy condition of the skin. Acne punctata, which may be accepted as synonymous with the above described comedone formation, occurs about puberty, and is a harmless condition which may remain without developing into true acne. Upon the supervention of inflammatory action the condition passes into that of acne vulgaris, in which the sebaceous gland and the surrounding connective tissue of the hair-follicle inflames, causing the appearance of a small hard papule, which may resolve without pus formation. More frequently, however, suppuration occurs, the papule becoming transformed into a circumscribed abscess or pustule, the condition then being recognised as *acne pustulosa*. Successive crops of papules appear, and many, but not necessarily all of these continue to suppurate in their turn, so that the duration of the disease is indefinitely prolonged. When the inflammatory action is severe and deeper, the papules assume the form of larger nodules of hard consistence and deep red or livid colour, this condition being recognised as *acne indurata*. In this latter there is sometimes no suppuration ; but more frequently after the discharge of a small amount of pus there remains a considerable degree of induration for a long time.

After the suppuration usually extensive scarring remains, often the face and shoulders being seamed by tracts of cicatricial tissue which may cause considerable unsightliness or disfigurement ; occasionally the skin between the shoulders in the site of these scars shows cheloid growths.

Whilst the advent of acne vulgaris is usually preceded by comedone formation, sometimes the inflammatory process attacks the apparently normal sebaceous glands. This is

seen in the acne papule which so commonly follows the administration of bromides and iodides, and which often ends in pustulation. A similar condition follows the application of skin stimulants like tar, in all of which instances the acne may appear in a patient at any age.

Diagnosis.—The site of the eruption is characteristic, though the rare form of *A. cachecticorum* may appear anywhere on the body, and the still rarer *A. varioloformis* attacks the forehead and scalp, leaving scars like small-pox.

The age—fourteen to thirty—the presence of comedones eventuating in pustules followed by scars and induration, the periodic liability to acute exacerbations following indiscretions in diet, and the marked greasy or oily condition of the skin distinguish it from all other eruptions.

Prognosis.—The natural tendency is for the eruption to disappear before twenty-five or thirty, and the amount of induration and scarring will depend mainly upon the depth of the pustules and the presence of the strumous diathesis.

Treatment.—The indications are for the correction of dietetic errors, anæmia, constipation, uterine disturbances, &c. The various local methods for the cleansing and disinfecting of the skin and for the removal of comedones and the preventing of pustulation will be found described in the fourth edition of the author's 'Dictionary of Treatment,' pp. 18–23. The main principle in the treatment of the comedones and of the true acne consists in the expression of the contents of the gland-ducts by pressure and friction after the free and vigorous use of soap and warm water. Any antiseptic ointment may then be rubbed into the skin of the affected region; the most valuable routine application being a weak ointment of sulphur, or of resorcin and bichloride of mercury, or a lotion containing precipitated sulphur and glycerin. Sulphur internally in small doses is often beneficial.

Under 'Phthisis' (Diagnosis and Treatment) Wright's method of the vaccine inoculation for that disease is described. This authority and his co-workers report excellent results in the treatment of acne when the method is carried out on similar lines. A little pus is taken from an acne spot, and the staphylococcus submitted to an incubator with suitable culture-medium. After the sterilisation of the culture the standardised emulsion is hypodermically injected, and the treatment in a few weeks is usually followed by at least marked amelioration of the disease. French states that there is no necessity for determination of the opsonic index; this shows a constant negative phase, as after minute tuberculin injections; neither is there any necessity to trouble about the particular staphylococcus, since by starting with the germ

from the acne pustule the specific organism, whether *S. aureus*, *albus*, or *citreus*, supplies the vaccine suitable in each case. The injections, each of which represents the sterilised products of 250,000,000 of the cocci, should be given about every seventeen or twenty-one days. No fever reaction or feeling of illness accompanies the treatment, which can be pursued as the patient follows his ordinary occupation. It is claimed for this method that disfigurement is prevented, and marked alleviation of the local inflammation procured—desiderata which are of special importance in the facial acne of female patients.

The condition formerly known as *Acne rosacea*, owing to the occasional presence of acne pustules from retained secretion of the sebaceous follicles, will be described under its proper title of 'Rosacea.'

ACROMEGALY

This is an affection also known as Marie's Disease, characterised by a hypertrophic enlargement chiefly of the bones and soft tissues of the face, feet, and hands, and almost invariably found in association with changes in the pituitary gland.

Etiology and Pathology.—The condition is more frequent in women; it appears most commonly about the age of thirty, rarely before twenty or after forty. The osseous system being chiefly involved has led to the suspicion of its rheumatoid origin, especially as rheumatic troubles have been known in some cases to precede it; but neither rheumatic, syphilitic, nor other known constitutional disorder can be said to possess any connection with the disease. The changes almost constantly found in the pituitary body after death from acromegaly have led to the general acceptance of the view that the condition is the direct result of a disturbance in the function of this gland. In almost every case it is considerably enlarged, and the hypertrophic growth in the region of the sella turcica is so great as often to cause serious pressure-symptoms from involvement of the optic nerves or chiasma. The gland is believed to manufacture an internal secretion whose action regulates the proportionate growth of different parts of the body as the internal thyroid secretion is believed to regulate certain metabolic processes. The excess of this pituitary secretion is consequently regarded as exhibiting itself in acromegaly, and according to some authorities occasionally in gigantism. The explanation of the condition of myxœdema is the opposite to this, the thyroid internal secretion being deficient. Those who regard exophthalmic goître as due to over-action of the thyroid, see an analogy in this disease to the one under consideration. Moreover, in acromegaly the

thyroid has been frequently found hypertrophied—according to some observers such has been the case in 50 per cent. of the examples of the disease—but sometimes it is atrophied or normal. The thymus gland is also frequently hypertrophied, but sometimes entirely absent.

Symptoms.—The disease begins insidiously. Occasionally there are vague pains and other sensory disturbances in the affected parts, which steadily enlarge until a most characteristic physiognomy and deformity become developed. The skull and its integuments increase in dimensions, but not usually proportionate to the enlargement of the bones and soft tissues of the face, which hypertrophy greatly, the bony markings being obviously exaggerated, especially about the orbits and zygomatic arches. The lower jaw hypertrophies more than the upper, causing a marked protrusion ; the lower lip keeps pace with the growth of the bone, and the teeth, not joining in the enlargement, are separated by the expansion of the alveolar septa.

The nose uniformly increases in size, the nostrils becoming broad and wide often to a greater extent than the ears, eyelids, and tongue, which also greatly hypertrophy. The disease is usually first observed in the hands and feet, which begin to grow coarse and spade-like, though the skin is normal, with its palmar and plantar grooves deepened. The increase in bulk of the giant-like feet and hands is seen under the Röntgen rays to be constituted by an hypertrophy of the bones, skin, subcutaneous tissues, and nails, though the latter are not unduly curved nor are the last phalanges bulbous as in other conditions. The great toe is often much more enlarged than the other portions of the foot, but seldom is a corresponding state of the thumb observable.

The clavicles, sternum, and ribs gain in bulk, and the cervical or dorsal vertebræ show some kyphosis. Sometimes the external genitals increase in size, and often wasting of the muscles has been observed. Rarely are the pelvic bones involved.

With these signs in advanced cases marked secondary symptoms may appear ; thus, owing to the enlargement of the larynx, palate, tonsils, tongue, and lips, the voice alters, and the vision fails from intracranial pressure upon the chiasma or optic nerves, bi-temporal hemianopia, or blindness from atrophy of the disc occurring. Headache, somnolency, and marked mental symptoms occasionally appear, the first mentioned symptom being often prominent and severe. The senses of taste and smell are often impaired. In some cases the hypertrophy of the thymus has been known to cause marked dulness over the upper part of the sternum, and cardiac disturbance, palpitation, dyspnœa, and asthmatic seizures may occur. A high degree of hypertrophy of the heart has been occasionally

observed. In some cases the thyroid enlargement is an obvious feature in the clinical picture during life. The disease runs a very chronic course, and sometimes the hypertrophic enlargement of the hands and feet may long remain evident before the face becomes involved.

Under the name of *micromegaly* the opposite condition has been described by Gilford and Hutchinson, in which immature and premature development of various parts of the body may be seen present at the same period.

Diagnosis.—The physiognomy is so characteristic that the affection cannot be mistaken when the signs have developed. In the early stages it may suggest *myxœdema*, but the natural condition of the hair and the absence of the harsh, dry state of the skin, and the absence of the slowness in speech, distinguish it from this disease. All the subjects of acromegaly appear to closely resemble each other, owing to the elongation of the face and the prognathus or projection of the lower jaw, so that, one case having been observed, the disease is afterwards recognised at a glance.

In *hypertrophic osteo-pulmonary arthropathy* there is observed a very chronic and slow enlargement of the feet and hands associated with hypertrophy of the long bones extending towards the elbows and knees. The last joints of the fingers and toes are greatly broadened and ridged, and shielded over by the long curved nails. The bones of the cranium and face are never involved, and the affection is nearly always associated with chronic pulmonary or pleural disease, though in a few cases it has been preceded by syphilis and cardiac affections.

Prognosis.—The disease is slowly progressive, though in some cases it may make a halt for many months, or even years. Death occurs in about twenty years from its first unmistakable development, the fatal result being generally caused by the cerebral tumour, by cardiac weakness, or by the advent of coma.

Treatment.—No drug has yet been found of any value in staying the slow progress of this disease, but intense headache may be relieved by full doses of antipyrin, exalgin, or other analgesic, and intracranial pressure may be sometimes lessened by large doses of iodides or by trephining. Thyroid, pituitary, thymus, lung, and other animal extracts have been tried in vain, and attempts at the removal of the diseased gland have hitherto proved fatal.

ACTINOMYCOSIS

This is a specific chronic infectious disease caused by the entrance into the body of a vegetable parasite—*Actinomyces bovis*, or the *ray fungus*.

Etiology and Pathology.—The disease is prevalent amongst horned cattle and horses, and occurs sometimes in pigs. In the human subject it is more common in Russia and Germany than in Great Britain; and men, owing to their greater chances of exposure, are affected much more frequently than women. Farmers, millers, grooms, coachmen, and stable-boys make up the great bulk of the victims of the disease. The actinomyces flourishes upon such cereals as rye, barley, and oats, and finds its way into the human body generally by the mouth, more frequently when the raw grain or straws are chewed than when the cereal is used as food in the ordinary way. It may lodge in the hollows of carious teeth and the crypts of the tonsils, or, being swallowed, may invade the abdominal organs. It has been known to frequently reach the respiratory tract by the inhaled air laden with the dust of the grain. Many instances are on record where the primary site of the disease has been found to be the skin, the parasite having apparently gained its entrance through minute abrasions caused by the sharp awns of the infected grain, splinters of wood, or stems of grass and cereals. It has been successfully inoculated into cattle, but there is no instance where the human disease has been traced to the consumption of the meat or milk of infected cows. The parasite has been successfully cultivated outside the body, and the virulence of the cultures experimentally demonstrated.

The organism is classed as a streptothrix bacterium, and more than one variety or species is suspected of producing lesions closely allied to those recognised as due to *Streptothrix Actinomyces*. The fungus, after gaining its entrance into the tissues, produces small slowly growing tumours caused by the irritation due to its presence; in the interior of these it increases and multiplies, forming granular spherical masses made up of minute colonies. These granules measure from about $\frac{1}{500}$ to $\frac{1}{250}$ inch in diameter and vary in colour from greyish white to greenish yellow or dark brown. As the inflammatory nodules soften, the pus, when examined after the addition of a little saline solution, will show the specific granule or actinomyces grain as a minute primrose-yellow speck. Under the microscope the granule is found to consist of a central core formed of closely interwoven branching threads or filaments and coccus-like spherical bodies. From this central mass of mycelium numerous threads radiate outwards, often showing a dichotomous method of branching. The circumferential part of the granule is occupied by the clubs, which are

minute flask- or pear-shaped bodies formed by the expansion of the distal end of each radiating mycelial thread. The younger granules are grey in colour and translucent, and do not show the club endings on their peripheries. These latter markings are better seen in human actinomycosis than in cultures, where their formation is always less distinct. True gonidia or spores grow freely on the surface of cultures; these are very resistant, and may preserve their vitality unimpaired for years.

The granules may be easily distinguished not only in the pus coming directly from the lesions, but also in the urine, sputum, fæces, and in the stroma of the affected tissue. They may be examined under a cover-glass with a drop of glycerin, and the threads or mycelial elements stain readily by Gram's method.

Pathological Anatomy.—The changes vary with the nature of the tissue invaded by the parasite. These in the main are such as are observable in the localised inflammatory processes which follow the introduction or inoculation of other irritating organisms into the tissues. A localised chronic progressive inflammation originates which causes destruction of the neighbouring cellular elements and invasion by leucocytes; granulation-tissue follows with the proliferation of the connective-tissue elements till a tumour is produced. This may assume large dimensions, as seen in the firm growths resembling malignant neoplasms which affect the lower jaw. Ultimately the new-growth breaks down and suppuration occurs, leaving, after the discharge of the pus, numerous chronically oozing sinuses and fistulæ. The disease spreads from one structure or organ to another by direct infiltration, or travelling by way of the lymphatics or blood-vessels it may invade organs at a distance from the original site of its introduction. When a soft organ like the liver is invaded it may become honeycombed by numerous suppurative foci which coalesce into large round masses surrounded by dense fibroid indurations. Adhesions form over the inflamed capsule, and the different organs in the abdomen may be slowly invaded as the parasite gains an entrance into the adjacent coils of bowel, or the pleural cavity may be reached through an extension from the diaphragm or lung.

Changes of a different kind are occasionally met with when the purulent collections are absorbed, true metastatic abscesses such as occur in pyæmia being found. This is only likely to occur when a secondary infection caused by pyogenic organisms takes place, but it appears proven that the parasite itself is pyogenic.

Symptoms.—These vary with the tissue, organs, or region invaded. The disease in the main possesses the features of a tubercular infection in its slow progressive development with

pyrexial symptoms and constitutional disturbances. During its later stages the clinical picture may be that of a chronic type of pyæmia, with its profound chills or rigors and sweatings.

The disease is most frequently found attacking the mouth, jaws, tongue, and neck, though any region may become involved after the fungus enters the blood-vessels. When the mouth has been the site of invasion, a hard tumour appears under the periosteum of the lower jaw, often opposite to carious teeth; this, at first resembling a sarcoma, slowly enlarges and finally softens, discharging pus containing the characteristic granules. Gradually the neighbouring tissues become involved and the growth may extend downwards in the neck involving the vertebræ or mediastinum, or upwards towards the skull infecting the base of the brain, the process simulating the progress of a tubercular necrosis of the soft and hard tissues.

In the abdomen and lung the symptoms are those of ulceration of the intestines with peritonitis, and of chronic bronchial or of interstitial pneumonia, to which the signs of multiple abscesses may be added. The clinical picture in these cases may closely resemble chronic tuberculosis, but as a rule the apices of the lung are free in pulmonary actinomycosis, the bases being chiefly affected, though pleuritis may supervene, and there is a tendency in the diseased process to attack the ribs and chest-wall. The multiple abscesses in the liver may lead to the production of symptoms of high fever and to the appearance of signs of hepatic suppuration, which, however, owing to its slow progress, may point in the direction of the abdominal walls or of the psoas muscle. The disease often appears in the region of the appendix.

When actinomycosis affects the brain the growth of the abscess and the progress of the case are more rapid, especially when the invasion is by the blood-vessels and not by the gradual extension from the base of the skull.

When primary inoculation of the skin by the awns of barley, rye, and oats, or splinters of wood, or stems of cereals occurs, soft tubercles of a livid, mottled, or yellowish colour appear; these enlarge, resemble a conglomeration of sarcomatous tumours covered over by a thin skin, which ulcerates, and the discharged secretion contains the characteristic granules.

Diagnosis.—This is generally impossible at first, but the clinical features of a chronic pyæmia should lead to an examination of the pus, sputum, urine, or fæces, and the detection of the characteristic ray fungus renders the diagnosis certain. The occupation of the patient should be noticed; farmers, grooms, and millers being most frequently affected.

Prognosis.—The disease tends to run a chronic course, and in those cases where it is localised the prognosis is good; but where suppuration occurs early and the blood-stream is

invaded, the course may be rapid and fatal from the formation of metastatic abscesses. Prognosis is better in lower- than in upper-jaw cases, and worse in thoracic than in abdominal cases.

Treatment.—The various surgical procedures for the local treatment of the disease are detailed in the author's 'Dictionary of Treatment,' pp. 23, 24. The internal administration of iodide of potassium has been proved to be of the greatest value, and in numerous cases the disease has yielded completely to doses of half a drachm three or four times daily. Recently, intravenous injections of collargol (2 per cent.) are advocated in thoracic and abdominal cases. The best results are obtained by the simultaneous administration of the iodide and by scraping, excision, or other surgical procedure; but the internal treatment must be persevered in for many months after the apparent eradication of the parasite, else relapses are certain to occur.

ADDISON'S DISEASE

This is a progressive malady characterised by profound general weakness, cardiac failure, gastric irritability, and increased pigmentation of the skin, and generally found associated with disease of the suprarenal capsules.

Etiology and Pathology.—It may occur at any period of life, but is rather more frequent in males of the labouring class at about the age of thirty. Trauma, grief, and mental exhaustion are regarded as exciting causes, and the disease is sometimes preceded by tubercular disease of the lung, vertebral bodies, or other bones.

The affection is believed to be the result of the absence of the adrenal secretion alone, or combined with the irritation of the abdominal sympathetic, which results from the destructive lesion in the glands. In the great majority of the cases the nature of the change in the adrenals is a fibro-caseous tubercular one, but simple atrophy, cancer, interstitial inflammation, and hæmorrhagic infarction have been found. That the disease is not simply due to adrenal inadequacy (as myxœdema is to loss of thyroid function) is rendered probable by the failure of adrenalin in removing the symptoms or curing the affection.

Symptoms.—The onset of the symptoms is so gradual as to generally escape notice for a time. The patient often complains of loss of muscular power, preceded by, accompanied with, or soon followed by nausea and vomiting.

1. The asthenia gradually becomes profound, and the loss of muscular and mental vigour produces a feeling of languor which, with the absence of marked emaciation and anæmia, is

often characteristic or highly suspicious. There is usually a slightly subnormal temperature.

2. The gastric symptoms begin in the presence of a clean moist tongue, with capriciousness or failure of appetite, often ending in complete anorexia. Nausea, which is generally present throughout the course of the disease, eventuates in vomiting and retching, which may end in death from pure exhaustion, or hiccough may hasten the end. Constipation is the rule, but obstinate diarrhœa sometimes supervenes. There is generally some muscular wasting in the later stages of the disease, but the subcutaneous fat is well marked to the end.

3. The heart shares in the general asthenia, the ventricular power steadily failing, so that the very compressible pulse can be only felt with difficulty; the blood-pressure is always low, and the tendency to syncopal attacks, palpitation, and breathlessness increases on the least exertion. Though the number of red corpuscles may show some diminution, signs of marked anæmia are absent.

4. Nervous symptoms are rarely prominent, vague pains in the loins and upper portion of the abdomen, headache, and slight disturbance of vision and hearing may be complained of; but in association with the stomach symptoms there may be marked pain in the epigastrium, which in the later stages may simulate peritonitis. The mind remains clear unless delirium or coma appears just before death.

5. The urine is free from albumin and sugar, generally there is no excess of pigment, and the specific gravity is lowered.

6. Pigmentation or bronzing is a sign of great importance, and though occasionally absent it is the feature which often suggests the presence of the disease and which usually settles the diagnosis. It may appear early or be delayed till near the termination of the affection. It may be seen to be an exaggeration of the normal skin-pigment (melanin), giving a dusky brownish-yellow colour not unlike that seen in the mulatto or Moor, but it is not uniformly distributed over the entire surface of the body. Thus it is most evident upon the exposed parts of the face, neck, and back of the hands and in those portions of the body which are naturally darker, as the anterior fold of the arm-pit, over the vertebral spines, the genitals and groins, and around the nipples. Where the skin has been exposed to irritation or pressure, as over the site of blisters and over the constriction of the waistband or garters in women and the braces in men, here the bronzing may be first noticed, and in advanced cases it is always best marked. The hair may be darkened, but the scalp, and palms of the hands, soles of the feet, and nails are unaffected. The bronzing is in the Malpighian layer, and though generally diffused there may often be found small very dark specks like moles

or deep sunburns on some parts where the discoloration is most intense.

A point of great diagnostic importance is the presence of pigmented blotches on the visible mucous membranes, as on the lips, inside of the cheeks and gums, and in the form of inky patches on the tongue and in the vagina. A minute examination may reveal some deposit of pigment in any of these places or upon the conjunctiva, which is generally pearly-white, and sometimes pigmentation has been revealed by the ophthalmoscope in the deeper structures of the eye.

Diagnosis.—There is little difficulty in advanced cases where the marked asthenia, vomiting, and characteristic pigmentation are present, but in the early stages the diagnosis is most difficult, especially in the absence of bronzing. Here the affection can only be recognised as present after excluding every other known cause of profound asthenia and gastric disturbance.

Pernicious anæmia is excluded by a careful examination of the blood.

Splenic anæmia is excluded by the absence of the enlarged spleen and by the absence of the blood-characteristics, though it must not be forgotten that the skin may be pigmented in splenic anæmia.

Cancer of the stomach may be excluded by the absence of marked emaciation and by the presence of free hydrochloric acid in the stomach-contents.

Bright's disease, in which constant vomiting is often associated with profound asthenia, is easily excluded by the examination of the urine and by attention to the pulse-tension.

☞ The physician must be upon his guard not to mistake the pigmentation which occurs in other affections for the bronzing characteristic of Addison's disease; this is only likely to occur by forgetting that in the latter case it is always associated with the profound asthenia, cardiac failure, and gastric irritability which produce its characteristic clinical picture. It is, however, well to note that pigmentation of the skin may be found associated with the following:

1. *Pregnancy*. Here the vomiting may also tend to mislead the physician; but the condition of the uterus and mammæ settles the diagnosis. The localised *chloasma* of uterine disease is never so uniformly diffused; the darkening is generally confined to the face, as is also the case in pregnancy.

2. The bronzing in *phtheiriasis* or lousiness constituting the so-called Vagabonds' Disease is easily diagnosed by the presence of the parasite on the clothing and the marks of the scratching, and the face is free from pigmentation.

3. *Tubercular*, *malignant*, or *lymphadenomatous growths* in the abdomen, especially when accompanied by peritonitis.

These are generally easily distinguished by the physical signs; and when *acanthosis nigricans* is met with in these malignant conditions the skin is rough and hypertrophied.

4. *Melanotic cancer* is distinguished by the presence of the primary tumour in the liver or elsewhere.

5. *Diabetes*, especially when associated with hepatic cirrhosis; here the presence of sugar settles the diagnosis.

6. *Phthisis.* 7. *Chronic syphilis.* 8. *Graves' disease.* 9. *Sclerodermia.* 10. *Pancreatic disease.* 11. *Malarial cachexia.* 12. *Pellagra.* 13. *Granular kidney* with valvular disease. 14. *Chronic gastric ulcer.* In all these cases the absence of Addison's disease is accepted by the presence of the physical signs and history of each affection.

15. *Jaundice*, easily for the most part diagnosed by the colour of the conjunctiva.

16. *Argyria.*—Here the leaden or slate colour is characteristic.

17. *Arsenical pigmentation.*—Generally, the history is sufficient to clear up this condition; if not, the presence of the drug in the urine may be sought for.

Prognosis.—The disease steadily progresses as a rule, though not seldom there are halts in its march for some months at a time. Death generally occurs inside two or three years, but occasionally it may be deferred for seven or more years. Acute cases have been reported which lasted only a few months, where death supervened before pigmentation occurred. It is very doubtful if recovery ever occurs. Death may occur at any stage from cardiac asthenia or collapse after diarrhœa or vomiting, or from hiccough.

Treatment.—In advanced cases rest in bed is essential owing to the danger of syncope. Symptoms of gastric disturbance call for bismuth and gastric sedatives, any anæmia may be corrected by iron and arsenic, and cardiac asthenia by strychnine hypodermically.

Suprarenal gland, its extract or active principle, has been freely tried, generally without any permanent effect, though many reporters affirm that it has a beneficial effect in raising the blood-pressure and preventing syncope.

Dose of the dry extract, 1 to 3 grs.; liquid extract, 10–15 mins.; adrenalin chloride solution (1 in 1,000) 10 to 30 mins. This latter can be administered hypodermically when diluted with saline solution.

ALBUMINURIA

This is the name given to the condition in which albumin is found in the urine in the form of serum-albumin and globulin, or their derivatives, acid- and alkali-albumins. Other proteids, as albumoses, peptones, fibrin, and nucleo-albumins, are met

with, and the conditions are known as albumosuria, peptonuria, &c.

Tests.—Three tests—heat, nitric acid, and picric acid—suffice for all ordinary clinical purposes. The cold nitric acid, indeed, will generally be found to answer all purposes, the others being occasionally used for confirmation in doubtful cases.

Heat.—If the urine is clear and acid it will only be necessary to fill a test-tube three parts full, and, holding it by the bottom end obliquely, to apply the flame of a spirit-lamp to the upper layer of the liquid. The heat throws down the serum-albumin and globulin (which both generally exist together) in the form of a white cloud, which slowly descends as a soft flaky precipitate. If phosphates be present a cloud will also form, which, however, is readily soluble on the addition of a few drops of acetic acid. If alkaline urine be heated in the above manner the albumin is not thrown down, because it exists in the form of alkali-albumin, which does not coagulate by heat; hence it is necessary to ensure acidity in alkaline or neutral urine by adding *a few drops* of acetic acid before heating. If, however, too much acid be added, the albumin is converted into acid-albumin, which also does not coagulate by heat, and this may be sometimes found to be the condition in highly acid urines or when a sample of normally acid urine chances to be poured into a test-tube which had a trace of nitric acid already remaining in it from a former testing. When the urine is turbid from urates, slight heat causes it to brighten, when further heating will coagulate the albumin; turbidity must be removed by filtration before testing.

Cold Nitric Acid (*Heller's test*).—Less than half an inch of the test-tube is filled by pure nitric acid; the tube is then held nearly horizontal and a few drachms of the urine are allowed to slowly trickle down the sides, or a pipette may be used to float it on the surface of the acid. Where the two fluids come in contact a white disc-shaped opacity marks the presence of albumin; some minutes may elapse before the disc appears, if only a trace of albumin be present. In this way 1 part of albumin in 50,000 of urine may be detected.

The presence of a faint diffuse haze near to the upper surface of the urine will indicate mucin, and a deeper cloud will indicate albumose: the former will not disappear on heating; the latter clears off, but returns on cooling.

A disc of minute crystals of urates forms in concentrated urine, which disappears on heating, and which does not form if the specimen be previously diluted with water.

In patients taking turpentine, copaiba, and sandalwood the acid causes an opacity which disappears on heating or upon the addition of alcohol. The coloured zone which often forms at the junction of the urine and acid may be disregarded when no opacity is present.

Picric Acid.—A cold saturated solution of picric acid, when gently poured into a test-tube half full of clear bright urine gives a whitish-yellow opacity which does not disappear on heating. If the urine be strongly alkaline the reaction does not occur. Picric acid also throws down alkaloids like quinine, antipyrin, and albumoses, but these disappear on heating.

The quantitative estimation of albumin is most readily determined by Esbach's albuminimeter, by which the depth of the precipitate produced by picric acid is measured in a graduated test-tube.

A rough estimate of the daily variations in albuminuria may be made by boiling the acidified urine in a test-tube and allowing it to settle, when the depth of the precipitate may be expressed in fractions of the entire column, as one-third, one-fourth, or one-eighth.

Causes of Albuminuria.—The *source* of the albumin is doubtless the albumin of the blood. This is proved by Aschoff, who found that upon injecting various proteids, the blood of the animal manufactured a 'precipitin' body which was always capable of precipitating *in vitro* the original proteid injected. The *cause* of albuminuria in renal disease is held to be a lesion of the glomerular epithelium, though the possibility of the proteid finding its way into the tubes by exudation is difficult of denial.

Excluding those instances where the presence of albumin is caused by pus and blood finding its way into the urine, cases of albuminuria may be divided into—

1. Those where there is obvious disease of the kidneys, as in Bright's disease, amyloid and fatty degeneration, renal tuberculosis, and acute or passive congestion.

2. Those cases where there is no marked renal lesion. This latter group will include :

(*a*) The so-called cyclic, postural, physiological, functional, or intermittent albuminuria, or albuminuria of adolescence.

(*b*) Febrile albuminuria, as seen in pneumonia, diphtheria, and the exanthemata, where the cause is supposed to depend upon some transient change in the glomerular epithelium, which passes off as the fever declines.

(*c*) Nervous albuminuria, as seen in head-injuries, epilepsy, convulsions, tetanus, and nervous excitement. The writer has noticed this in numerous candidates coming direct for physical examination after the ordeal of a public-service competitive examination lasting for several hours. In these latter cases often the albumin never returned.

(*d*) Hæmic albuminuria, as seen in anæmias, diabetes, scurvy, purpura, leukæmia, ulcerative endocarditis, and the chronic poisoning by lead, mercury, and syphilis.

Only the cyclic or functional variety may be further mentioned here as the other forms can best be studied under their

respective headings. This condition is of importance in relation to life insurance, and diverse views regarding its signification are held, some maintaining that it is a purely physiological phenomenon, others that its presence indicates a pathological change in the renal epithelium liable to be followed by the establishment of permanent kidney disease.

The various synonyms already mentioned convey its chief characteristics or the symptoms of its different varieties. Occurring most frequently in young males, the albumin, generally slight in amount, is usually absent in the urine passed on arising from bed in the morning, hence it is often spoken of as orthostatic or postural albuminuria. The albumin returns after breakfast, after prolonged exertion, or bathing, the chief characteristic being its paroxysmal, intermittent, or cyclic appearance. Save for the albuminuria, the urine is otherwise normal in colour, amount, and density; occasionally a few hyaline casts may be found, but these are often noticed as present in healthy subjects, when the specimen has been submitted to centrifugalisation, who have not had albuminuria. The other signs and symptoms of renal disease are of course absent.

There is one variety of this transient albuminuria so markedly dependent upon the ingestion of food that it is spoken of as dietetic albuminuria. It appears after eating largely of proteid food (eggs, &c.), but sometimes after a full meal of any kind.

Diagnosis.—The periodic appearance of the albumin in the otherwise healthy urine, and especially the absence of it in the morning specimen, the normal state of cardiac tension and the age of the subject, serve to settle the diagnosis, only however, after repeated examinations of the urine. Nucleo-albumin or mucin, which has no clinical significance, must not be mistaken for albumin; it is not precipitated by the heat test, and when the cold nitric test is employed the haze does not appear in the line of contact, but towards the upper surface of the urine.

Prognosis.—In the large majority of cases complete recovery ensues, the albumin permanently disappearing; a small minority of the cases are believed to pass into one of the forms of Bright's disease, so that some insurance authorities refuse all albuminurics. As a rule, however, where the condition rapidly responds to rest and diet, and the albumin remains absent for a few weeks, the prognosis is certainly favourable.

Treatment.—Moderate rest in the orthostatic variety, and strict attention to the correction of all dietetic errors and dyspeptic symptoms, as a rule, will be all that is necessary in most cases.

ALBUMOSURIA

The presence of albumoses in the urine (hetero- and deutero-albumose) has little clinical importance. They are recognised by the picric and nitric acid tests, and have been found associated with chronic suppuration, cancer, and the puerperal state. Their presence is, however, marked as a clinical feature in multiple myelomata, where the hetero-albumose (Bence-Jones's albumose) is claimed to be distinctive, giving a black reaction with caustic soda and lead acetate. Albumosuria is also claimed to be a distinctive feature in cerebro-spinal meningitis, though absent in tubercular meningitis.

ALCOHOLISM, ACUTE

It is unnecessary to describe the various and variable symptoms preceding the final stage of acute alcoholic poisoning, as these are unfortunately too well known. Coma supervenes after one large poisonous draught, or after a series of small doses swallowed at short intervals.

Diagnosis.—The recognition of alcoholic coma is a matter of vital importance in many cases. The skin is cold, though generally somewhat cyanosed, the limbs are flaccid, the pupils are dilated, the pulse full, and the breathing deep or stertorous. The temperature is subnormal or markedly reduced ; sometimes muscular tremors may be noticed. When the coma is complete the patient cannot be roused, and all evidence of sensation is lost, as in chloroform narcosis. The breath smells strongly of alcohol, though this should not be relied upon where a history of the case is impossible, as spirits are so commonly resorted to upon the onset of every serious seizure. Death takes place from paralysis of the respiratory centre.

From *post-epileptic* coma, the history of the case when available is valuable, and the tongue should be carefully examined, the presence of an injury to this organ indicating a previous convulsion. From the coma produced by *other narcotic poisons* the only conclusive evidence will be found by an examination of the stomach-contents ; but the odour of the breath is important, as chloroform, chloral, laudanum, and other narcotics can be recognised. The drug may be detected in the urine ; morphia can in this way be demonstrated by the iodic acid or by the diazo-reaction. The pupils in opium-poisoning are contracted to a pin's point. In the coma of *cerebral hæmorrhage* or *embolism* the stertor is generally pronounced, the pupils vary and are often unequal, but are generally contracted, especially when the pons is involved, and some difference in the flaccidity of the limbs on the opposite sides of the body may reveal hemiplegia. The presence of a

scalp-wound may reveal other evidence of *head-injury*. *Diabetic* coma is generally associated with much sugar in the urine ; the presence of acetone is not of much value in the diagnosis. *Uræmic* coma is as a rule easily diagnosed by the presence of a large amount of albumin in the urine ; but it must not be forgotten that this may be present after large cerebral hæmorrhages and after first attacks of epilepsy, and it may only exist in small amount in the urine of the fibroid kidney. It is therefore very important to look for œdema and to examine for cardiac hypertrophy.

Prognosis.—This will depend upon the amount of alcohol taken and upon the length of time between its administration and the active measures undertaken for its treatment. Recovery may sometimes follow after twelve hours of absolute coma, if treatment be persisted in.

Treatment.—The contents of the stomach should be immediately removed by the syphon or stomach-pump, or, failing these, by the hypodermic injection of $\frac{1}{10}$ gr. apomorphine. The organ should be washed out with copious quantities of very warm water, and finally filled with a strong hot infusion of coffee ; heat to the extremities, and to the general cutaneous surface, counter-irritation, galvanism, and a full hypodermic dose of strychnine ($\frac{1}{10}$ gr.) may be all indicated in serious cases.

ALCOHOLISM, CHRONIC

This is a disease of the nervous system induced by chronic indulgence in alcoholic excesses, characterised by a variety of mental, nervous, and physical symptoms, amongst which the diminution of will-power to resist the appetite for alcohol is the most marked pathological feature. In long-established examples of the disease, every organ and tissue of the body give evidence of functional and structural alteration, though the patient may never have experienced the full physiological action of the drug as demonstrated by the group of symptoms called drunkenness.

Owing to the retarded metabolism, obesity is often a marked sign in the earlier stages, whilst at a late stage emaciation may result from gastric disturbance.

The nervous system suffers as a rule early, and one of the most important signs is tremor, beginning in the hands and feet. It is most marked in the morning and passes off after eating or after a dose of alcohol ; the tongue and lips are also often involved, and the sign is only noticeable on some delicate muscular movement, as an attempt at writing or forcible protrusion of the tongue. At first it can be controlled by volition, but gradually it gets beyond the influence of the will, and fine muscular combined movements become more or less

permanently affected, and spasms supervene. The peripheral nervous system gradually becomes involved, and various types of neuritis develop, causing tenderness, pain, and motor paralysis (see under 'Neuritis'). Epilepsy is not uncommon. Mental failure or torpor, loss of memory and of will-power, headache, insomnia, noises in the ears, irritability of temper, and melancholia gradually appear. The moral sense is generally seriously affected along with the intellectual, and it may be accepted that the victim rarely if ever tells the truth about the amount of alcohol which he usually imbibes. Prominent amongst these symptoms of cerebral degeneration stand out the craving for alcohol and the inability of the will-power to resist it, and the deceitful methods which he pursues to obtain his supply. The digestive system suffers markedly; dyspepsia, nausea, morning vomiting, and loathing at all food are generally well marked; the tongue is foul, and tremulous and flabby, though sometimes red and irritable; the catarrhal gastritis extends to the duodenum and bowels, causing sometimes slight jaundice and diarrhœa. The liver swells and becomes tender, and finally, if the poison be taken in a concentrated form and when fasting, hepatic cirrhosis supervenes. The facial expression is often characteristic, and the enlargement of the small vessels in the skin over the nose and cheeks, approaching to the condition of rosacea, renders the diagnosis often tolerably certain before any history is obtained. Serious vascular changes are always present, the heart-muscle degenerates and the ventricle dilates, fatty degeneration and fibroid thickening of the arterial walls are common, and renal changes appear. Delirium tremens may appear at any stage, and the alcoholic subject is more susceptible to most diseases, and, when affected, owing to his weak resisting power he is more liable to succumb to disease and injury than those who do not partake of alcohol.

Diagnosis.—The best plan for the physician to pursue is to look out for the symptoms of nerve and digestive disorders which are characteristic of the affection, and to make no inquiries about the patient's habits as regards alcohol till he has satisfied himself about the general condition of the organs and tissues. In this way he obtains more trustworthy information by not placing the patient upon his guard. Thus a frank history of morning vomiting or nausea, insomnia, loss of appetite, slight hallucinations of hearing, and the early signs and symptoms of peripheral neuritis may be elicited easily sometimes from an alcoholic patient who may not himself suspect the cause of his trouble.

The investigation of the tremor is of primary importance: it must not be confounded with that of *disseminated c. s. sclerosis*, which is absent when the patient is in complete repose, and which is only brought into action when he attempts

to catch hold of some object. Unlike the fine shaking movements of alcoholism, it is a coarse oscillating intention tremor exaggerated in proportion to the muscular co-ordination required by the effort attempted. In chronic alcoholism there is no nystagmus nor scanning speech.

The tremor of *paralysis agitans* has a regular rapid rhythm with limited range which is characteristic.

Mercurial tremor resembles the last mentioned; it may be distinguished from the shaking of alcoholism by the presence of other mercurial symptoms, by the history, and by the fact that it may persist during sleep.

The coarse purposeless movements of *chorea* can scarcely be mistaken for alcoholic tremor. When they simulate it, however, their distribution and the history of the case render the diagnosis easy.

Hysterical tremors, coarse and fine, are more jerky, varying, and irregular, and are always associated with other emotional motor phenomena; they are also more or less purposive.

The *tremor of senility* is only seen in those of advanced age; its range of movement is very limited, and the head is nearly always affected.

Simple tremor without any known cause is liable to be mistaken for alcoholic tremor, but its constant character and permanence, and its liability to affect the head and facial muscles, and the absence of all other symptoms, are pathognomonic.

The *nervous tremor* noticeable in sensitive persons under examination lacks the chief characteristics of the alcoholic, and passes completely away after the transient emotional excitement subsides.

General paralysis of the insane, especially where the mental symptoms are latent, closely resembles chronic alcoholism, and the diagnosis can only be determined by the presence of morning sickness, cardiac, hepatic, vascular, and renal changes, the absence of irregularity in the pupils, and the temporary improvement in alcoholism while the patient rigidly abstains. In general paralysis the disease is progressive, and more rapid and uninfluenced by any treatment. The peripheral neuritis of chronic alcoholism may be mistaken for *locomotor ataxia*, especially if the gastric symptoms are frequent and prominent. The absence of the Argyll-Robertson pupil, the presence of tenderness of the calf-muscles, and the pointing of the toes or dropping of the foot, are characteristic of the pseudo-ataxic form of multiple neuritis. The dyspepsia of chronic alcoholism is easily differentiated from other gastric ailments if discreetly investigated, the patient often stating that no relief is obtainable from the discomfort and nausea except by taking alcohol. In arriving at a diagnosis, the physician must ever remember that the alcohol habit may be grafted upon another diseased

condition which may have been also the cause of the habit, as general paralysis, insomnia, dementia, &c.

Prognosis.—When the affection is established, this is always serious. Much, if not all, depends upon the amount of will-power left. The writer's experience is that in those chronic cases where the patient glibly affirms that he can easily leave off alcohol there is *always* a bad ending. The longer that the patient can be kept from alcohol the greater the hope of a permanent reformation; and when one year's abstinence has expired his prospects are good, and after double this period there is every hope of a permanent cure, provided always that he remains a strict teetotaler. The prognosis is grave in patients with an hereditary history of alcoholism or insanity, and in those whose moral sense was below the average standard prior to the establishment of the alcohol habit. The prognosis is good as regards the disappearance of insomnia, gastritis, cardiac disturbance and hepatic enlargement, and even neuritis, if the patient rigidly abstains.

Treatment.—The writer has in the 'Dictionary of Treatment' (4th ed.), pp. 27–32, stated his conviction that the danger of the establishment of the alcohol habit is enormously increased by the practice of taking alcohol in concentrated form, and that preventive treatment based on this consideration would speedily alter the aspect of the great drink-evil if alcohol could be procurable only of the claret or sherry strength. The various remedies for the relief of the symptoms of chronic alcoholism are given in detail in the pages referred to, and need not be here repeated. The first question to be settled is the serious one of the sudden or gradual withdrawal of the poison, and there need be no hesitation in adopting the former method and immediately stopping supplies.

It is generally found impossible to carry out for the necessary time prohibitory measures in ordinary domestic life, and there can be no doubt about the wisdom of insisting upon the patient entering for a period of at least six months a good inebriate asylum. The value of the so-called 'gold' cure, hypnotic suggestion, and other methods is yet to be demonstrated. Though the benefit of hypodermics of strychnine, alone or with atropine, is beyond doubt, it must be recognised that no drug-treatment is of any permanent use unless measures be at the same time taken for the elevation of the moral tone of the victim, and especially for assisting his weakened will-power or volitional defect. No hope must be held out that the cured or reformed individual can ever return to the habit of using alcohol, even in smallest quantities. His only hope can rest upon absolute abstinence for the remainder of his life.

ALOPECIA

Under this title are included all cases of baldness. They are generally classed as four—viz. Congenital, Senile, Premature, and the special condition known as Alopecia areata.

The *congenital* variety is very rare, and when present is generally associated with arrest of development of the nails; it may be universal over the entire skin, or only partial, and as the growth of the body proceeds the hair may appear on some portions of the skin, or it may become normal over the usual hairy regions.

Senile alopecia, so common in males of advanced age, is but a part of the general failure in nutrition and consequent wasting of all the elements of the cutaneous tissue. It usually begins at the vertex and spreads towards the temples and the occiput till the entire scalp is stripped, but generally a zone of variable depth is left at the sides and below the occipital protuberance.

Premature alopecia appears generally about the age of twenty-five, but may sometimes be noticed earlier. It begins in a progressive thinning of the hair over the sides of the frontal region, which extends backwards; soon the vertex becomes affected, and the shedding from this area progresses forwards till the entire anterior portion of the scalp becomes denuded. Finally a fringe like that seen in the senile form is all that is left. The progress is generally slow, and unless active treatment arrests the shedding permanent baldness results. The generally accepted view of the pathology of this form of alopecia is that it is the direct result of *seborrhœa oleosa* or of *seborrhœa sicca* (dandriff); in the former the scalp is covered with a yellow oily or waxy secretion, and in the latter it seems powdered over with fine glistening scales. (See under 'Seborrhœa,' in Vol. II.)

Sabouraud has demonstrated that the causal seborrhœa is produced by a micro-bacillus which he has succeeded in cultivating; this finds its way into the orifice of the hair-follicle, ultimately destroying the papilla by the pressure of its cocoons. Heredity and other influences only tell in so far as they favour or retard the growth of the specific bacillus.

Diagnosis.—Premature baldness can scarcely be confounded with any other affection; but since the shedding of the hair which follows profound constitutional causes, as in fevers, myxœdema, diabetes, syphilis, phthisis, &c., is usually included under this name, it is essential that each be differentiated by an examination of the history and symptoms of the primary affection. In the same manner alopecia caused by the local ravages of favus, ringworm, erysipelas, psoriasis, lupus, eczema, and other lesions is for the most part easily differentiated. The rare cases following severe and prolonged mental emotion are also easily recognised.

Prognosis.—This will depend upon the cause; it is hopeful in the seborrhœic form only when this is detected in its early stages and when treatment of the seborrhœa is promptly and most tenaciously adhered to. Most cases falling under the physician's notice are beyond cure, when the scalp is already well stripped. The baldness following fevers and many of the conditions just mentioned are easily cured by treatment of the constitutional condition. The results of thyroid administration in the baldness of myxœdema are generally most striking. The alopecia left after favus is often permanent; baldness is rarely extensive or permanent save in the worst cases of ringworm.

Treatment.—The chief indication is the management of the seborrhœa, and for this anti-parasitic remedies are essential. Ointments are better than lotions, especially in the dry form, and they should be well rubbed in after cleansing with borax. Mercurials are amongst the most reliable, and the ung. hyd. ammon. is the best; next comes the dilute ung. hyd. nit. Lotions containing the perchloride of mercury, or resorcin, or sulphur, or sozo-iodolate of soda are recommended; the reader will find numerous formulæ upon pages 71 and 72 of the author's 'Dictionary of Treatment.' Cantharides at every stage is useful especially as a mild stimulant to the hair-bulbs after fevers and other temporary causes of alopecia.

ALOPECIA AREATA

This singular affection begins in a circular patch of complete baldness, usually upon the scalp at its posterior or lateral aspects, in size from that of a shilling to a crown-piece, and of a smooth ivory whiteness. Other spots may form, and by coalescing a large portion or the entire scalp be denuded, and the eyelashes and eyebrows may be shed; the face and indeed the entire surface of the body have been seen to become completely hairless in some cases.

While the disease is progressing the hair is easily pulled out beyond the margin of the patch, and always a number of stumps of hair shaped like a point of exclamation are to be found at the circumference of the bald circle. These latter, according to Crocker, are of great diagnostic importance, and they also show that the disease is spreading; each consists of a portion of the normal hair-shaft with its withered root. As the patch ceases to grow these clubbed stumps disappear; fine colourless hair forms over its surface, which gradually becomes pigmented, and a cure spontaneously occurs in a few months. In some cases the baldness is permanent, but recovery may occur after a few years of waiting.

Sometimes a band of complete baldness may extend from ear to ear over the occiput.

Pathology.—Though this form of baldness differs widely in appearance from the ordinary seborrhœic variety previously described, Sabouraud maintains that in all cases occurring after puberty it is caused by the same parasite, and that it is contagious. Other authorities insist upon the affection being a trophoneurotic one, and there are rare varieties of it where this origin seems certain. Crocker believes that it has some relationship with ringworm. Hallopeau has recently maintained that its parasitic origin is proven by its occurrence in families, barracks, and schools, where numerous individuals are massed together in conditions where the head is kept uncovered, by its multiple areas of distribution, by its clinical features, and by its yielding to anti-parasitic agents.

Diagnosis.—The only difficulty is in the exclusion of ordinary *ringworm*. This is generally easy if the club-shaped hair-stumps be found at the margin of the alopecia; these come out with the least touch, and they are quite distinct from the ragged, broken, and twisted hairs which are found scattered all over the patch of ringworm. The smooth billiard-ball appearance of the entire patch is generally so characteristic as to preclude a mistake, and the microscopic examination settles the matter by demonstrating the absence of fungus in alopecia and its presence in ringworm.

Prognosis.—In young subjects, where the club-shaped stumps have ceased to be visible, the prognosis is always good, but in older patients with rapid coalescence of many spots, and where the scalp seems to be withered and wrinkled and the hair-follicles wasted, the hope of recovery is very slender, especially if the disease has lasted for several years. Where the hair has been rapidly shed over the entire body the prognosis is very bad; it is good in the very circumscribed forms following neuralgia, neuritis, nerve-injury, or severe mental emotion.

Treatment.—The writer's routine is to rub in strong sulphurous acid daily by means of lint on a probe, and after a few weeks to apply strong cantharidine applications so as to produce mild vesication. Sabouraud uses sulphur ointment, and upon page 73 of the author's 'Dictionary of Treatment' numerous formulæ and methods will be found mentioned. It is, however, probable that most of the recoveries attributed to the light treatment and to various agents employed are examples of the spontaneous tendency of the disease to recovery, and support the neurotic theory of its etiology.

ANÆMIA

With our imperfect knowledge of the changes which take place in the blood, both in health and disease, an accurate definition of anæmia is not possible at present. The main clinical features by which we recognise the condition are those obviously caused by poverty of the red corpuscles, especially by their deficiency in hæmoglobin or by their diminution in number. The symptoms of bloodlessness are obvious and common to anæmias produced by the different causes: pallor of the skin and visible mucous membranes, palpitation of the heart and shortness of breath on slight exertion, languor and tendency to syncope, with slight œdema; emaciation is absent unless some wasting disease is present. The heart is generally somewhat dilated, various murmurs are audible, and in women the menstrual function is in abeyance. Anæmias are classified as Primary and Secondary. In the primary forms the lesion is not traceable to disease of any organ or to the escape of blood from the vessels, but it is believed to be the result of some change in the blood itself, or to some diseased action in some portion of the organism not yet capable of demonstration.

The primary anæmias, described under their own headings, are Chlorosis, Pernicious Anæmia, Splenic Anæmia, and the different forms of Leukæmia, though in these latter it is the white blood-corpuscles which give the characteristics to the blood-examination, and by some they are classified as secondary.

Secondary anæmia owes its origin most frequently to—

1. Hæmorrhage from injury or disease, as in gastric or intestinal ulcer, uterine, renal, bladder, or other affection in which there is a breach of surface; the profound anæmia produced by ankylostoma may be regarded as a hæmorrhage.

2. It may arise from the blood-exhaustion produced by chronic suppuration, albuminuria, and the growth of malignant tumours.

3. From inanition caused by want of food, as in starvation or occlusion of the gullet, or by failure of the digestive process, as in gastric diseases and intestinal parasitic affections.

4. From the direct destruction of the red corpuscles by the malarial parasite.

5. From the action of poisons like lead, arsenic, and mercury, or toxins like those of syphilis, malaria, and the exanthemata, or the poison produced by the decomposition of bothriocephalus.

Diagnosis.—The examination of the blood in secondary anæmia, as in a case of severe recent hæmorrhage, reveals some degree of hydræmia owing to the rapid absorption of

lymph ; there is increase of white cells (leucocytosis) and the increase is in the polymorphonuclear neutrophiles, whilst the small uninuclear lymphocytes are diminished.

The red cells are diminished in number ; they are generally smaller and contain less hæmoglobin than in the normal condition ; they are paler, and some poikilocytosis is noticeable. Nucleated red cells or erythroblasts of the normal red-cell size (normoblasts) from the marrow are present. The number of red cells rapidly increases and the leucocytosis speedily diminishes, but the rise in the hæmoglobin is a much slower process.

ANÆMIA, PERNICIOUS

This disease is also called Idiopathic or Progressive Pernicious Anæmia.

Etiology.—Its causation and pathology are obscure ; the most probable theory is that the disease is the result of toxic *hæmolysis* induced by poisons produced in the intestinal tract. This is supported by the presence of large amounts of iron in the liver, urobilin in the urine, and the frequency with which oral, gastric, or intestinal lesions are noticeable. No micro-organism has been detected in the blood, and the changes found in the bone-marrow are now believed to be secondary phenomena and not the cause of what was supposed to be a defective *hæmogenesis*. That this latter process plays a *rôle* in the disease is proven by the faulty shapes of the corpuscular blood-elements and by the increase in the red marrow, while the yellow marrow is diminished. Stockman's theory that the condition is the result of a series of small recurring hæmorrhages can only cover a small percentage of the cases. Hunter maintains that the toxæmia resulting from the absorption of the poisons by the intestinal mucosa has its origin in a specific streptococcic infection which is directly due to oral sepsis caused by necrosing teeth, stomatitis, or glossitis, the result of sewer-gas poisoning. Middle-aged males and females constitute the bulk of the cases, though it may occur in childhood and old age. The anæmia sometimes follows a period of prolonged mental strain or worry, or appears after a sudden fright, or after pregnancy, parturition, or lactation ; and the disease is more common in rural districts than in cities.

Symptoms.—These come on slowly and insidiously, the anæmia showing itself by a pallor usually of a pale lemon colour resembling slight jaundice ; the breathing and heart are disturbed on the least exertion, dyspnœa, palpitation, and syncopal attacks supervening ; weariness, languor, and extreme debility are felt, and they are often the first symptoms of

the disease. The visible mucous membranes become blanched, and though there may be some wasting of muscle, the subcutaneous fat does not diminish, giving the patient a flabby waxen look. The pulse is quick, the heart is dilated, and murmurs are heard over the pulmonary area, at the apex, and over the jugular vein near to the sternum; the pupils dilate; noises in the ears, gastric disturbances, from anorexia to obstinate vomiting with diarrhœa, supervene. Small hæmorrhages into the skin or from the mucous surfaces are common, and the retina in most cases demonstrates their presence by flame-shaped stains in the vicinity of the optic disc. The temperature is generally normal, but most cases at some period in their course show marked, but moderate, pyrexia. The urine may be dark from urobilin, but otherwise it is normal. The liver slightly enlarges, but the spleen and lymphatic glands are generally normal, and there is often some œdema of the legs. Transient paralysis and mental symptoms sometimes occur which have been attributed to small hæmorrhages into the cord or brain.

Examination of the blood reveals important diagnostic changes; when a drop exudes from a needle-prick in the lobule of the ear or in the finger, the eye detects its extreme watery condition. Under the microscope the number of red corpuscles may be found by the Thomas-Zeiss or Gowers' hæmocytometer to be considerably under 1,000,000 instead of the usual 5,000,000 in each cubic millimetre of blood. The cells do not form rouleaux; they vary in size and shape, and this poikilocytosis is always well marked. The pear, horse-shoe, or oval form of the corpuscles is striking, and the majority of them are larger than normal (megalocytes). Red corpuscles of much smaller size are also present (microcytes). Nucleated red cells are to be found, which, according to their sizes, have been named normoblasts, megaloblasts, and microblasts. The presence of the megaloblasts is, however, of most importance in the diagnosis, and in stained films they must be carefully searched for. Some of the blasts stain readily and present a granular appearance, and the blood-platelets are scanty, or absent altogether.

The white corpuscles are diminished in number except in the last stages, but the differential count shows that there is an increase in the smaller or lymphocyte variety at the expense of the polymorphonuclear neutrophiles. Occasionally marrow-cells or myelocytes may be found.

These changes are only to be made out in stained preparations made in the laboratory. The physician has no difficulty in procuring a specimen for examination by allowing a drop of blood to fall upon the end of a microscope-slide or bit of glass, and by drawing this along the entire width and length of the slide by means of a piece of smooth tissue-paper an even

layer of corpuscles is obtained which can be afterwards stained, and examined at leisure.

A point of greatest importance in the examination of the fresh blood is the determination of the hæmoglobin percentage. The hæmoglobin is always reduced, but never reduced in proportion to the fall in the corpuscles. It is not uncommon, for example, to find the latter reduced to 1,000,000—which is only one-fifth the normal number—whilst the reduction in the hæmoglobin may only amount to one-third of the normal. This relation of the percentage of hæmoglobin to the number of red corpuscles is called the 'colour-index,' and the colour-index is one of the diagnostic methods by which pernicious anæmia is distinguished from chlorosis and the secondary anæmias, as it is always higher than the normal in the affection under consideration. Thus, suppose, as just instanced, the red corpuscles only count out 1,000,000, a number equivalent to 20 per cent. of the normal, and should the amount of hæmoglobin as determined by the hæmoglobinometer be found to be 30 per cent. of the normal, the colour-index is expressed as $\frac{30}{20}$ or 1·5, the normal colour-index being 1. In other words, each individual corpuscle is larger, or contains a greater quantity of hæmoglobin, than in health, and this always maintains in advanced cases of the disease.

Morbid Anatomy.—The pathological anatomical changes are such as may be expected in profound anæmia from whatever cause—viz. extreme fatty degeneration of the heart and small vessels, less so of the liver and kidney, and lymphoid alterations in the bone-marrow, with the presence of Ehrlich's gigantoblasts. Hunter maintains that the deposition of iron in the form of hæmosiderin in the outer and middle zones of the hepatic lobule is distinctive, and this iron derivative of hæmoglobin is also found in the spleen and kidney. Lorrain Smith has shown that the total volume of the watery blood may be increased. The stomach is wasted, and marked atrophic changes are usually found in the secreting glandular apparatus, though the general body-fat remains as a rule little diminished in amount.

Diagnosis.—This can only be arrived at after the exclusion of every recognised form of secondary anæmia. The most difficult disease to exclude is *cancer*, especially of the stomach or bowel. In malignant disease the anæmia seldom is so profound, the fall in red cells rarely reaches 50 per cent., and the hæmoglobin fall is always greater than the corpuscular decline; the other characters of the blood before described are absent in cancer, and moreover the history, age of the patient, emaciation, and local signs generally settle the diagnosis. The same remarks apply to *syphilis* in its chronic stages.

Chlorosis is excluded by the blood-examination. In this condition the hæmoglobin is always very low, much lower

in proportion to the corpuscular loss; in other words, the colour-index is below the normal, poikilocytes are few, nucleated red corpuscles (erythroblasts), if present, are of the size of the normal cells (normoblasts), megaloblasts are seldom found, megalocytes and microcytes are few. The age, sex of the patient, normal colour of the urine, and the rapid response to iron as a rule demonstrate chlorosis in the absence of blood-examination. *Bothriocephalus anæmia* cannot be distinguished from the pernicious variety by the blood-examination: here the diagnosis can only be settled by the discovery of the parasites in the fæces.

From the anæmia of *hæmorrhage*, pernicious anæmia may be distinguished by the history, by the high colour-index, and by the absence of leucocytosis, and by the other blood-characters. *Splenic anæmia* is excluded by the absence of enlarged spleen, and *leukæmia* by the absence of the great increase in the number of leucocytes and of marrow-cells (myelocytes) or lymphocytes.

Chronic malaria, *plumbism*, *purpura*, and other forms of severe secondary anæmia, conform to the type of chlorotic blood with its low colour-index.

Prognosis.—This is always grave, and the rapidity of the progress of the case may be inferred by the presence of the following: (1) The *progressive* type of the anæmia—*i.e.* the absence of remissions; (2) the existence of a high colour-index; (3) of megaloblasts; (4) of megalocytes; (5) of lymphocytosis; and (6) of a count of under 1,000,000. It is to be suspected that many of the so-called cures are cases of secondary anæmias; nothing is so common in this strange affection as the temporary remission and apparent cure which is so soon followed by relapse and death. The disease may prove fatal in a few months, or it may be extended in remission cases for two or more years; recovery, though rare, is certainly possible.

Treatment.—The only drug of any value is arsenic. This may be given in small doses—2 minims of Fowler's solution—which should be steadily increased till ten times this amount be taken. Where the gastric irritability prevents oral administration, the solution (made without lavender) may be given hypodermically when well diluted with water, or it may be administered by the rectum. Sometimes arsenious acid is tolerated in such cases when given by the mouth in the form of a keratin-coated pill. Cacodylate of sodium (1 grain) may be given hypodermically; though the experiments of Fraser prove that this drug does not yield up its arsenium in the body, many still believe that it is a valuable agent. It has not, however, answered the high expectations which earlier reports raised. Its hypodermic administration should be stopped upon the tenth day, and not resumed for a week. Antistreptococcic serum has recently been credited with

special curative virtues. Iron at a later stage may be tried if the symptoms yield to arsenic. Red marrow, intestinal antiseptics, and lavage, transfusion, and other methods of treatment will be found detailed in the author's 'Dictionary of Treatment,' pp. 41–46.

ANÆMIA, SPLENIC

This is a disease characterised by a progressive anæmia, with enlargement of the spleen and without marked leucocytosis.

Etiology.—Nothing is known definitely about the causation of this condition, which usually affects adult males. A microbic and a toxæmic theory are held as in pernicious anæmia, and some regard the anæmia as the result of the splenic enlargement.

Symptoms.—In addition to the symptoms of profound anæmia described under the last heading, the patient experiences attacks of severe pain in the splenic region, with moderate pyrexia and repeated bleeding, especially from the nose. The enlargement of the spleen is progressive, and the organ may weigh up to seven pounds. There is some increase in the size of the liver, but the lymphatic glands are not enlarged, and emaciation only occurs towards the end in cases of long duration. The Malpighian bodies are found to be greatly atrophied, the fibrous tissue much increased, and the pulp wasted. The disease is very rare, chiefly attacking males after the time of puberty ; it runs a slow and protracted course for many years, but ultimately ends fatally, though death has occurred in six months in some cases.

The blood-examination reveals loss of red corpuscles, which may fall to nearly 1,000,000, but as a rule the reduction is not so great as in pernicious anæmia. The hæmoglobin reduction is always greater than the corpuscular deficiency, hence this anæmia is classed under the chlorotic type. The cells are smaller than the normal (microcytes are present), but there are few, if any, poikilocytes. Leucocytes are only transiently increased after attacks of fever or hæmorrhage, so that leucocytosis is not a feature of this anæmia ; when it does occur, it is the small uninuclear lymphocytes which are slightly increased. The pathology of the disease is obscure, and no micro-organism has been found ; and the part played by the splenic enlargement is doubtful, though this has been sometimes considered as the primary lesion, and hence the disease has been described as Primary Splenomegaly.

Diagnosis.—From *pernicious anæmia* this rare disease is generally easily differentiated by the presence of the great splenic enlargement, the blood-examination, the generally longer duration, and the greater tendency to hæmorrhages.

From *splenic leukæmia* it is easily differentiated by the absence in the blood of myelocytes and by the absence of the great excess of the polymorphonuclear cells.

From *cirrhosis of the liver*, *malarial*, *tubercular*, *syphilitic*, and *malignant diseases of the spleen* the clinical features of these affections and the blood-character generally are sufficient to establish a diagnosis. When cirrhosis of the liver supervenes upon splenic anæmia the combination is known as Banti's disease, and some observers regard this latter disease as hereditary splenic anæmia. (See under 'Spleen.')

Splenic anæmia differs from *Hodgkin's disease* in the absence of the characteristic enlargement of the lymphatic glands, in the more severe type of the anæmia and in the slighter degree of hepatic enlargement.

Prognosis.—The disease may be regarded as always fatal, though it may last for as long as twenty years; but even in the milder types death may at any stage be caused by a severe hæmorrhage or syncopal attack. A few recoveries have been reported after splenectomy. In the later states of the disease the supervention of hepatic cirrhosis, which is frequent, constitutes a grave condition, dropsy and jaundice following, the condition being then practically identical with Banti's disease.

Treatment.—All the remedies indicated in pernicious anæmia have proved valueless, but removal of the spleen should be resorted to with some hope of success if the anæmia has not been permitted to proceed too far.

ANCHYLOSTOMIASIS

This is a diseased condition characterised by a profound anæmia caused by the parasite *Anchylostomum* or *Ankylostomum duodenale*.

Etiology.—This nematode worm is found in most of the warm parts of the earth, and has recently been imported into the north and south of England by miners. It has been always common in the miners employed in tunnelling in Italy and Egypt and India. In America it has been named *Uncinaria Americana*. The male measures one-third and the female nearly two-thirds of an inch. It inhabits the jejunum more commonly than the duodenum, where it holds on to the mucous membrane by its suctorial mouth and teeth, gorging itself with blood, extracting and passing through its alimentary canal enormous numbers of red discs and white corpuscles, and probably secreting a toxin which increases the anæmia. The encapsuled ova find their way into the intestine in muddy drinking-water, or in the clay or soil which adheres to the fingers or gets accidentally mixed with food. Loos has shown that the larvæ may penetrate the skin, reach

the air-vesicles of the lung *via* the blood or lymph, and from there enter the bronchi and trachea, pass out of the glottis to the gullet, and become sexually mature in the duodenum. The symptoms are those of a profound anæmia of the chlorotic type, and the blood-examination gives the same characters. Dyspeptic troubles and abdominal pain may be prominent features in some cases. Death results from exhaustion after a variable duration of many months or years, if proper treatment be not resorted to. Occasionally, when there is only one infection, the disease has been known to disappear owing to the gradual dying out of the generations of the parasite after several years.

Diagnosis.—This is practically impossible from an examination of the symptoms and blood only. There is always, however, great increase in the eosinophile-cells. Fortunately the ova are easily found in the fæces, where they exist in great numbers. A minute portion of bowel-matter is smeared over a slide, a drop of water added, and a cover applied. Under a medium power the eggs are seen to be oval, ·060 mm., covered with a transparent thin shell (devoid of an operculum), through which the segmentation of the yolk is easily seen. From the ova of *ascaris* which are unsegmented and have a gelatinous envelope, and from those of *oxyuris* which are about the same size but unsymmetrical, being flatter on one side than on the other, and which contain a visible embryo, the anchylostomum-egg is easily differentiated. The ova of *trichocephalus* have a clear spot at each end of their thick brown oval shell. The *larvæ* of *Anguillula stercoralis* are often found in association with the *ova* of anchylostoma in freshly passed fæces.

Prognosis.—This is always good after the expulsion of the parasites has been effected, but occasionally the worm penetrates the mucosa and lies between it and the muscular layer, from which position it is dislodged with difficulty.

Treatment.—All the usual anthelmintics are now discarded for thymol. After a twelve hours' fast following a mild purge, 30 grs. (2 grms.) thymol may be given in cachet form every two hours for three times. No oil, alcohol, butter, or strong alkali should be given along with or after the drug, lest the thymol might be absorbed. A purge will be found to bring away the dead parasites in great numbers. If ova appear inside a week, the treatment must be repeated. Iron, arsenic, and other agents should be administered for the anæmia after the expulsion of the worms.

Prophylaxis is obvious: the most rigid cleanliness is all that is required. Boycott and Haldane have shown from a study of the parasite in the Cornish mines that the ova and freshly hatched larvæ are not infective, but that if encapsuled larvæ are swallowed the ova appear in the fæces after about thirty days. This 'encapsuled' stage is reached in three or four weeks

from the first hatching of the eggs in water, after which the larva moults once or twice in its chitinous sheath.

ANEURISM

This may be defined as a localised dilatation of an artery forming a tumour containing fluid or solid blood, and whose sac is composed of one or more of the coats originally constituting the arterial wall or of the tissues in the immediate neighbourhood. This definition therefore includes the *diffused* aneurism, whose sac contains no traces of arterial wall; the *fusiform*, whose sac may consist of a uniform dilatation of the three walls of the artery; also the *sacculated*, whose tunic may consist of one or more of the arterial coats, forming a pouch on one aspect of the vessel with which it communicates by a narrow neck; and the *dissecting* aneurism, which consists of a space between the arterial coats caused by rupture and escape of blood. The old terms *false* and *true* as applied to aneurisms are obsolete, as no large aneurisms contain in their sac all three of the original coats, which constitution formerly entitled them to be called 'true.' Indeed, the sac may in many cases be regarded as strictly devoid of any of the original vascular coats, being formed of a layer of fibrous material produced by inflammatory or reparative action in the coats.

Etiology.—It most frequently appears at about thirty-five to forty years, and in males, and herein lies the clue to the etiology and pathology of aneurism. It occurs at an age when strong muscular strain overlaps or supervenes upon the beginning of atheromatous degeneration. At a later stage the degeneration has increased, but the muscular power is weakened so that violent muscular effort becomes a lessening quantity. The chief causes which lead to arterial changes at an early age are (1) increased blood-pressure from *continuous* strain and from *sudden* or intermittent strain, (2) syphilis, (3) chronic alcoholism and over-eating, and (4) renal disease.

Other causes of aneurism are to be found in subjects with healthy vessels, as surgical injury to the arterial wall, embolism, and micro-organisms.

Symptoms.—The chief sign common to all aneurisms is the presence of an expansile pulsating tumour in the line of a large artery, with a systolic thrill and murmur which diminish on pressing the vessel on the proximal side. Pressure-symptoms are often present; they are caused by the stretching, erosion, or compression of nerve-trunks in the vicinity of the tumour, or by the pressure of the tumour on veins or hollow viscera.

The subject is mainly surgical, and only aneurism of the aorta need be here referred to.

Aneurism of the Thoracic Aorta.—Often in the early stages the symptoms are entirely latent, and they vary with the portion of the arch affected, the size and variety of the tumour, whether this be a saccular or a uniform dilatation, and with the direction in which the growth extends. There is often a history of syphilis, of prolonged heavy muscular work or sudden strain, with evidence of degenerative changes in the radial or other accessible vessel. The subjects are generally strong muscular males of about the age of forty. Owing to the position of the tumour, the diagnosis may in some instances depend entirely upon the symptoms, no signs being present. Pressure-symptoms or pressure-signs are of greatest importance.

The *ascending* portion is the most frequently involved, and it is not unusual to find this uniformly dilated, and the consequent stretching of the aortic ring leads to aortic regurgitation, with the usual after-consequences of cardiac hypertrophy on the left side. As a rule the physical *signs* are best marked when this portion of the aorta is affected; and the pressure-*symptoms* are most in evidence when the transverse arch is involved. The only evidence, however, of serious dilatation of the ascending portion may be cardiac pain and angina with a systolic aortic bruit.

Small sacculated aneurisms, just above the sinuses of Valsalva may, however, produce no signs or symptoms till they cause a fatal rupture into the pericardium. If a little higher up, the tumour will grow forwards, pulsation appearing in the second or third intercostal space, generally on the right side, and erosion of the ribs and sternum follows. Springing from the convex border of the ascending portion, it may, instead of growing forwards, encroach upon the right pleura, producing lung-symptoms, and compress the superior vena cava and cause œdema of the head and arms. If very large, it may dislocate the heart and cause pressure upon the right recurrent laryngeal nerve and inferior vena cava. Where a moderate-sized tumour springs from the concave aspect of the ascending portion, it may reach the surface to the left of the sternum, having pressed upon the pulmonary artery and embarrassed the right heart. Rupture may occur into the pericardium, pleura, or externally. The pulsation in such a tumour is characteristic: it is synchronous with the cardiac systole and it is expansile; there is often a thrill to be felt, and commonly a systolic but very rarely a diastolic murmur to be heard, and there is dulness on percussion over the swelling. There is generally accentuation of the second sound, but it is a noteworthy fact that hypertrophy of the heart very seldom, if ever, occurs in aortic aneurism, except, as already mentioned, when the ascending portion is uniformly dilated, involving the valves, or where a small sacculated aneurism implicates the valve in the sinus of Valsalva.

In aneurism of the *transverse* portion the effects of pressure upon the neighbouring structures may be the only symptoms to guide the physician to a diagnosis unless the tumour spring from the convex summit of the arch, where it may form a pulsating tumour projecting behind the sternum upwards into the neck and involving the innominate, left carotid, or even the subclavian, and ultimately eroding the sternum. The most common direction for an aneurism of this portion of the aorta to take is backwards, where its extension causes erosion of the vertebræ and pressure upon the trachea, bronchus, pneumogastric nerve, pulmonary artery, sympathetic, œsophagus, thoracic duct, or intercostal nerves.

Pressure upon the recurrent laryngeal produces dyspnœa, with stridor and paroxysmal fits of croupy or clanging cough ; abductor paralysis of one vocal cord, generally the left, is demonstrable by the laryngoscope, and after the supervention of paralytic contracture of the adductor the bovine character of the cough is especially distinctive.

Pressure on the trachea and bronchus causes difficulty in breathing, with profuse thin expectoration, which ultimately becomes bloody from ulceration. The air may be prevented from entering the left lung by the obstruction of the bronchus, and the respiratory murmur is then weakened or absent.

Tracheal tugging is a sign of great importance, and it is claimed to be distinctive of deep-seated aneurismal pressure on the left bronchus. It is elicited by placing the patient in the sitting posture with his mouth closed and his chin elevated ; the physician, standing behind him, grasps the cricoid cartilage and presses it upwards with the tips of both index-fingers ; as the aorta becomes distended the aneurism pushes the bronchus downwards before it, and the bronchus drags the trachea along with it at each cardiac pulsation. This sign is not detected in cases where solid tumours press upon the bronchus.

Pressure upon the sympathetic causes dilatation of the pupil at first, after which it generally remains contracted from paralysis, but it may vary ; nevertheless, inequality of the pupils should always be looked for as a sign of some value. Inequality of the radial pulses indicates an aneurism or tumour compressing the innominate or subclavian on the side upon which the pulse is small, and a large thoracic aneurism may obliterate the pulsation in the femorals.

Aneurism of the *descending* portion of the arch may erode the bodies of the adjoining vertebræ, injuring the spinal cord and intercostal nerves, and producing a pulsating tumour in the scapular region accompanied by intense pain, dysphagia from compression of the œsophagus, bronchiectasis with retained secretion and fever from pressure on the bronchus, and compression of the lung, or pneumonia. Rupture into the

pleural cavity commonly occurs, but sometimes the sac discharges into the œsophagus.

A similar train of symptoms is produced by aneurisms involving the descending thoracic aorta.

As the disease progresses, the pressure causes such marked pain and distress that the patient becomes emaciated and wears out from exhaustion often before the sac ruptures.

Diagnosis.—The early recognition of thoracic aneurism is difficult and often impossible. Suspicion should be aroused in cases of anomalous ailments referred to the upper part of the thorax by finding evidence of presenility in the arteries of the upper extremity, especially if there be a history of syphilis and prolonged muscular exertion, or of severe strain in conjunction with alcoholism.

When pulsation is visible it is generally to the right of the sternum, most frequently in the second interspace ; by careful inspection and palpation its *expansile* nature should be made out. In this way the pulsation produced by a solid tumour lying in contact with an artery is excluded. Unfortunately this important point is difficult unless the fingers can be so applied to the circumference of the swelling that the lateral expansion can be recognised. Pye-Smith's method is helpful. He uses a piece of plaster with a vertical slit in its centre ; on being stuck over the tumour the slit may be observed to widen on each pulsation. Pulsation can only be felt in some cases by placing one hand on the front and the other over the patient's spine. A thrill may often be made out (which is sometimes diastolic) when it strikes the hand with a jar or shock ; it may be preceded by a systolic vibration, or this may exist alone.

Dulness on percussion, with a feeling of increased resistance, can generally be elicited over the tumour unless it be small and deep-seated. By careful percussion in many instances the origin of the tumour from the ascending portion of the arch can be made out from the dulness being confined to the right of the manubrium. Small saccular aneurisms may elicit distinctive dulness between the second right interspace and the left border of the sternum.

Auscultation will often reveal the cardiac sounds, especially the second, much accentuated and clanging. A systolic murmur is common, but not of much significance unless its characters convey the conviction that it is produced in the sac. Where the aneurism is close to the sinus of Valsalva, involving the aortic valve, the characteristic diastolic bruit is always present. Often no adventitious sound is discernible.

The interpretation of the pressure-symptoms is of great diagnostic value ; the phenomenon of tracheal tugging has been already mentioned. The feebleness of the respiratory sounds over the left lung may reveal pressure on the left bronchus, which

may be the only evidence of the disease, but generally paroxysmal cough and dyspnœa are also present. The characters of the clanging cough and laryngeal symptoms caused by pressure on the recurrent laryngeal have been already mentioned, and their presence is of great importance. Especially as sudden stridor with dyspnœa coming on in severe paroxysms may be the first indication of the gravity of the situation, the differentiation of the symptoms caused by pressure upon the trachea and upon the recurrent nerve is necessary, as the dyspnœa caused by the former is not relieved by tracheotomy.

Pain caused by aneurismal pressure is to be carefully investigated. It is of two kinds: that produced by the irritation which the pressure induces in sensory nerves is liable to be severe and to come on in more or less violent and irregular paroxysms, whilst the steady gnawing pain caused by the osseous erosion is also often characteristic. The former pain may extend from the precordial region down the arms and closely simulate angina pectoris. Sir William Gairdner pointed out the beneficial effects of large doses of iodide of potassium in relieving aneurismal pain, and suggested that a therapeutic diagnosis might be arrived at by watching its effects.

Hæmorrhage is a sign of much value, and hæmoptysis in the early stages should arouse suspicion. The source of the bleeding may be the ulceration caused by the pressure on the trachea, bronchus, or lung, in which cases it may be frequent and slight in amount or it may be caused by rupture of the sac, which is generally but not always fatal at the time. The presence of recurrent hæmoptysis, with dyspnœa and clubbing of the fingers, all of which are not uncommon in aneurism, may lead to an erroneous diagnosis of phthisis.

Inequality of the radial pulses is a diagnostic sign of some importance, and the sphygmograph may be used for corroboration of the digital examination. On the affected or weakened side the tracing shows a slanting upstroke with flat apex, no tidal wave, and little, if any, dicrotism.

Venous congestion produced by pressure on the vena cava has been already referred to: it may amount to deep cyanosis, with dropsy of the neck, face, and one or both arms. There may be a loud continuous murmur develop, which would show that the sac had opened into the vein.

The pupillary signs require careful investigation. At first, from the irritation produced by aneurisms of the transverse portion, the pupil will dilate; but as paralysis occurs it contracts, owing to the destruction of the cilio-spinal branches, and there may be flushing of the face and neck.

The greatest difficulty in diagnosis will be the exclusion of solid malignant mediastinal tumours. The history of these, with the early emaciation, enlargement of glands, the escape of the arteries in the general pressure, the marked involvement

of the veins, and the often shorter duration of the new-growth, will settle the diagnosis. The presence of tracheal tugging is strongly in favour of aneurismal tumour, as is also the relief afforded to pain by rest and iodides.

The absence of the strong diastolic shock and of the expansile nature of the pulsation when mediastinal tumours pulsate are strong negative features in mediastinal growths.

The Röntgen rays should be pressed into the service in all doubtful cases.

Prognosis.—This is always most grave, but life can often be prolonged by careful treatment for many years, and in a very small percentage of cases complete obliteration of the aneurism may be effected. Death is generally sudden; but many cases die from the exhaustion caused by the pressure and erosion before the sac ruptures, and not a few die from some other disease.

Treatment.—Owing to the position of the tumour, the ordinary surgical procedures are impossible. The only hope lies in the methods which tend to produce coagulation within the sac, and these are only likely to succeed in saccular aneurisms communicating by a narrow orifice with the aorta.

The introduction of foreign bodies, as coils of fine wire, horsehair, needles, &c., has generally led to disasters. Galvano-puncture or electrolysis has in a few instances assisted the deposition of layers of fibrin so that complete consolidation has resulted; but there can be little doubt that in the majority of cases death has been rapidly hastened by such treatment. Macewen's plan seems more rational. He inserts one or more pins into the tumour, and moves them about so that their points can be made to touch the opposite wall of the sac and produce irritation which sets up reparative exudation, causing the formation of white thrombi in organic connection with the inner wall of the aneurism.

Notwithstanding the evidence of a few successes following the above-mentioned surgical procedures, it may be affirmed that the only justifiable line of practice is to be found in medical treatment.

The main element in this is rest. Valsalva, who first introduced this method, insisted upon numerous blood-lettings, which, however, were generally omitted by Tufnell and Bellingham, who afterwards modified it in various details.

The patient is kept in bed for a period varying from two to three months, and on no account permitted to sit up even for an instant, though he may be allowed to turn occasionally from side to side with slowness and deliberation, the object being by starvation to so reduce the amount of blood and the cardiac force as to greatly lessen the pressure upon the aneurismal wall, after which, by judicious feeding, the blood

is to be slowly enriched with the view of causing the deposit of laminated fibrin in the sac. In plethoric subjects frequent small bleedings may be necessary. The diet is to be carefully restricted. The total allowance of solid food is not to exceed 10 oz. daily, which may consist of well-cooked meat or fish and biscuit, and liquid nourishment to the amount of 10 oz. of milk is allowed. Bellingham's dietary consists of bread and butter for breakfast, 2 oz. ; meat and bread for dinner, 2 oz. each ; bread for supper, 2 oz. ; with 2 oz. milk at each meal. As a rule stimulants are forbidden. The bowels are carefully regulated, occasional salines being given, and complete repose of body and mind as far as possible secured. Pain is to be relieved by opium when it interferes with rest and sleep.

Of drugs which may be used alone or as adjuvants to the rest cure there cannot be any question as to the great value of iodide of potassium. This relieves aneurismal pain in a very remarkable manner, and it is generally believed to have some effect in promoting coagulation within the sac, and also in lowering the vascular tension so that the pressure is reduced in the sac. The writer has dissected a specimen of saccular thoracic aneurism which entirely consolidated under the above treatment; death was caused by phthisis after the tumour had become solid by the deposition of layers of fibrin. Thirty grains (2 grms.) should be given three or four times a day.

Chloride of calcium in doses of 15–30 grs. (1 to 2 grms.), with the view of markedly increasing the coagulability of the blood, has been advocated by Sir A. Wright ; and the author, in his ' Dictionary of Treatment,' p. 47, has recorded a case under his care where apparent cure resulted in an aneurism of the aorta (abdominal) from the use of this drug.

Hypodermic injections of sterilised 2 per cent. solutions of pure gelatin have been credited with successes, but tetanus and other untoward results are also reported. Protonuclein is also said to be valuable.

(For the details of other methods the reader is referred to pages 46 to 49 of the ' Dictionary of Treatment.')

Aneurism of the abdominal aorta, though suspected far more frequently than thoracic aneurism, is a much rarer affection. The presence of a pulsating tumour in the epigastric region is common in malignant disease of the pyloric end of the stomach, of the pancreas, of the left hepatic lobe, or of the gall-bladder. The tumour, in these cases, when grasped by the fingers is found not to be expansile ; it does not expand laterally—*i.e.* the thumb and fingers are not thrust apart by each pulsation as in aneurism. By placing the patient upon his knees and elbows, the tumour is lifted off the pulsating aorta and the vibration ceases to be felt by the hand. The pulsation of the aorta so commonly seen and felt in neurotic

women and in anæmic patients is never expansile, and is easily recognised, though a systolic murmur may be present. Aneurisms of the abdominal aorta do not descend with the movement of the diaphragm as other tumours do.

The symptoms are those produced by the pressure of the tumour, and erosion of the vertebræ may cause severe paroxysmal neuralgic pain radiating to the sides or shooting down the legs, and paraplegia, preceded by numbness and tingling, may supervene. The distinctive characters of the tumour are its median position in the vicinity of the cœliac axis, its expansile nature, its saccular or fusiform shape, and the presence of a systolic murmur. The femoral pulse may be retarded and weakened. Gastric symptoms as vomiting, intestinal troubles as obstinate constipation, and hepatic signs as jaundice, may result from pressure. The tumour may project into the flanks or loins or towards the thorax, and rupture may take place into the pleura, intestine, retroperitoneal tissues, or into the peritoneum.

The causation, prognosis, and treatment, discussed under Aneurism of the Thoracic Aorta, apply to cases of abdominal aneurism. A few cases of obliteration of the sac by the deposition of layers of fibrin caused by cessation of the circulation through digital or instrumental pressure have been recorded. This dangerous procedure, however, is only possible when the tumour is a considerable distance from the diaphragm.

ANGINA PECTORIS

This is a form of cardiac oppression or embarrassment coming on suddenly with intense radiating pain, generally accompanied by a feeling of impending death and often ending in a fatal issue.

Etiology and Pathology.—The true or grave variety of the disease appears after the later portion of middle life (forty-five to fifty-five years), chiefly in males engaged in mental occupations during some muscular exertion, as in hill-climbing, swimming, or running ; its first appearance may be caused by severe mental emotion, and it is very liable to return on every recurrence of its original exciting cause. There are still many points to be cleared up about its pathology, and little advantage accrues from the numerous classifications of the malady. It will suffice for clinical purposes to consider true angina as practically always associated with grave disease of the heart and vessels ; whilst the false or pseudo-angina may be regarded as a vaso-motor phenomenon, the result of altered innervation independent of cardio-vascular lesions. In the true or grave form the cardiac affection may be fatty infiltration or degeneration, aortic regurgitation or stenosis, mitral stenosis, aortic

atheroma, or adherent pericardium, but the characteristic lesion in the gravest cases of primary angina is *disease of the coronary arteries*. The explanation of the production of the symptoms witnessed during an anginal attack is difficult, as probably many factors are at work in the formation of the clinical picture. There is probably an acute obstruction in the cardiac circulation from spasm originating in the diseased and partly occluded coronary arteries ; temporary anæmia of the heart-muscle follows, with acute dilatation and increased blood-pressure in the ventricular cavities. In other cases a neuralgia or neurosis affecting the cardiac plexus is believed to induce either a state of muscular spasm or the opposite condition of paralysis, which is rapidly followed by acute ventricular dilatation. In the pseudo-anginal attack, which may be practically regarded as never fatal, the main or only factor appears to be a disturbance of the vaso-motor centres which produces a sudden contraction of the coats of all the arteries, small and large. This causes a rapid rise in the arterial blood-pressure which the ventricular walls being unable to cope with, acute distension or dilatation supervenes.

It is more than probable that general vaso-motor spasm is often superadded to the conditions which prevail in the grave or true angina caused by coronary or other structural disease of the heart, since in many cases, but not in all, there is obvious increased arterial tension, though the distension of the heart often found after death is not associated with rigidity of the muscular walls, the spasm having passed off.

The severe agonising pain experienced during the attack is usually accepted as being the direct result of the increased mechanical pressure to which the nerves are submitted in the cardiac walls and in the endo- and peri-cardial investiture. In this connection Carter has drawn attention to the changes which may be often detected during life in the cardiac walls after the attack has passed off. Endocarditis, pericarditis, and dilatation, as evidenced by increase in the area of dulness, and sometimes a transient anasarca are observable.

Symptoms.—The attack comes on suddenly during some exertion or upon exposure to cold. The pain is intense and agonising, originating in the precordial region, with a sense of constriction, as if the heart and thorax were being forcibly compressed ; it radiates back towards the scapula, up to the left shoulder, and down the left arm, though sometimes it may be most acutely felt on the right side ; tingling and numbness often accompany the pain. The face is pallid and covered with cold perspiration ; the lips are livid, though cyanosis is absent ; the expression is indicative of anguish and dread ; the attitude of the patient is one of immobility, clutching some fixed support within his reach ; and the breathing is restrained or suspended.

The condition of the heart during the attack may be apparently normal and regular; not infrequently the pulse may be found little altered, in marked contrast with the symptoms of impending death. It is generally harder than normal, but it may be thready and irregular, though not increased in frequency. The attack lasts but a few minutes, and in some cases a few seconds, passing off as rapidly as it came; but it may return again at short intervals for one or two hours, though a recurrence may not take place for months or years. A free secretion of colourless urine and a noisy expulsion of gas from the stomach often occur as the paroxysm passes off. The attack leaves the patient prostrated and weak for some hours. Death may occur in the first or any subsequent attack, or from the onset of syncope without the development of anginal symptoms.

Diagnosis.—The most important point is the elimination of false or *pseudo-angina*. This is most frequent in neurotic women, and may come on periodically when the patient is in repose; it occurs at an earlier age and in association with other nerve-symptoms; the attacks last longer, and the patient generally may be noticed to move about in a restless excitable state, which affords a marked contrast to the immobile posture of the true angina. There is generally no evidence of organic heart-disease, and there is often a history of toxic causes, as tobacco-smoking and excessive tea-drinking, and the usual signs and causes of arterial degeneration are absent, as atheroma, syphilis, alcoholism, habitual strain, &c. In the majority of cases of pseudo-angina there are marked vaso-motor symptoms, as deficient circulation in the hands and feet, and often the arterial tension is high between the attacks without any organic cause, and there is often an emotional cause for the attack, or a severe dyspepsia. True anginal seizures can usually be differentiated from attacks of *nephritic* and *hepatic colic* by a careful observation of the site of the pain and the direction in which it radiates. The presence of *aneurism* may be generally negatived by the absence of pressure symptoms and signs; *acute pleuritis* and *pleurodynia* by the less sudden onset, longer duration, and the signs peculiar to these affections. *Hysteria* usually demonstrates its presence by the globus, by the emotional phenomena, and the restless excitement which overshadow the purely cardiac symptoms which may be present.

Prognosis.—This is always good in pseudo-angina and always most grave in the true affection. Where the attacks recur with increasing severity and upon slight exertion in an elderly patient the subject of diseased vessels and with an aortic lesion and hypertrophied heart, death is almost certain to occur in an attack or during a syncopal seizure. Nevertheless life may be prolonged in considerable comfort by suitable treatment even in the most unpromising cases. The

development of regurgitation at the mitral orifice sometimes mitigates or prevents the anginal seizure.

Treatment.—This consists (1) in the exhibition of agents for the relief of the attacks, and (2) the use of such measures between the attacks as may tend to prevent their recurrence.

Nitrite of amyl, by causing rapid dilatation of the blood-vessels, generally gives almost immediate relief. Some authorities believe that it acts by removing cramp of the ventricular walls. The best form for its administration is the glass capsule (5 minims each), which, when nipped between the finger and thumb, permits of the contents being inhaled through the nose. Several may be used, as there is practically no danger from its administration. It often fails to relieve pseudo-angina. Nitroglycerin ($\frac{1}{100}$ grain) in tabloid form acts with less rapidity. Nitrite of soda, erythrol tetranitrate, and other vaso-dilators may be also used, but as a rule these should be reserved for cases where the patient has sufficient warning of his attack to permit time for their absorption. Waugh advises the subcutaneous injection of small doses of atropine every ten minutes in conjunction with the nitroglycerin treatment, with the intention of prolonging the action of the latter drug.

Chloroform inhalation or morphia hypodermically may be resorted to in those rare cases where nitrites fail, and oxygen may give much relief. Dyspeptic complications may call for emetics; hysterical or neurotic symptoms may be relieved by valerian, ether, or asafetida. Heat to the extremities in vaso-motor types of the disease may give relief. Absolute rest in bed should be insisted upon for a few days after the attack has passed off, with the view of permitting the cardiac walls to regain their normal tone.

In the intervals between the attacks attention must be paid to every known cause which tends to bring them about. Exercise must be most cautiously indulged in, and everything which tends to increase vascular tension must be avoided, as sudden exertion after a meal. The only allowable muscular exertion is slow and gentle walking on the level. Worry, brain-fag, irregular hours, and excitement of all kinds are to be avoided. Tobacco, alcohol, tea, and coffee must be forbidden in the toxic cases, and over-eating in all cases.

Upon the theory that the anginal attack is a pure neuralgia, nervine tonics have been advocated, but with the exception of arsenic they are of little service.

Iodides unquestionably afford the best treatment between the attacks; 30 grs. (2 grms.) may be given during the day, and in severe cases the daily dose may reach double this amount and may be taken for years with short intermissions. The writer finds that it may be combined to great advantage with arsenic for short periods.

Any intercurrent heart-lesion will demand its appropriate treatment; and upon the whole strophanthus with strychnine is safer than digitalis because of the risk of increasing blood-pressure. As a rule, cardiac tonics should be prescribed with nitroglycerin to avoid peripheral contraction; they can be combined with the iodides.

In those cases where attacks are frequent, they may be successfully warded off for long periods by the writer's method of directing the patient to break up the B.P. tabloid of nitroglycerin into six or eight fragments and take one every half-hour during the day as he goes about his usual occupation. In this way the vascular tension may be kept slightly reduced for long periods without any discomfort or ill effect. (See 'Dictionary of Treatment,' 4th ed. pp. 48-52.)

ANKYLOSTOMIASIS. (See ANCHYLOSTOMIASIS.)

ANTHRAX. (See MALIGNANT PUSTULE.)

APHASIA

This is the name given to various defects of speech and writing produced by disease of the higher cerebral speech-centres, though Bastian still insists that the term should be confined to the symptoms produced by disease of Broca's convolution.

Anarthria is the term used to designate defects of articulation or difficulty of utterance caused by bulbar paralysis, involving the lower speech-centres; this must be carefully separated from aphasia, as must also be *Aphonia*, or mere voicelessness.

The subject of aphasia is a rather complex one, owing to the different varieties of the affection being sometimes represented in any given case. The usual classification of the speech-defect under consideration is into motor aphasia and sensory aphasia.

Under the term 'higher speech-centres,' four are included:

1. The motor speech-centre in Broca's convolution—the posterior part of the left third frontal. In this area are stored the memories of the co-ordinated movements of the lips, tongue, palate, and larynx which are necessary for articulate speech; hence it is frequently spoken of as the glosso-kinæsthetic centre.

2. The auditory speech-centre, situated about the posterior half or third of the left upper temporo-sphenoidal convolution.

In this region the memory of the sounds of words is registered or stored up.

3. The visual speech-centre, located in the left angular gyrus and part of the supra-marginal gyrus, in which are stored up the memories of the forms of written or printed words.

4. The motor writing-centre, supposed to be located in the posterior third of the left second frontal convolution, in which is stored up the memory of the delicate movements of the hand required for writing. It is known as the cheiro-kinæsthetic centre.

These centres are connected by commissural fibres, and are all located in the left cerebral hemisphere in right-handed people, and in the right hemisphere in those who habitually use the left hand. Hence ordinary motor aphasia is very generally found associated with right-sided hemiplegia. The centres are in the neighbourhood of the Sylvian fissure and derive their blood-supply from the middle cerebral artery. The lesion may be hæmorrhagic, embolic, thrombotic; or it may be caused by tumour or abscess, and in rare cases it may be functional. It may involve only one centre or a portion of one centre, or the damage may affect two or more centres more or less completely. In order to interpret the different phenomena met with in aphasia, it is necessary to remember the progress of the development of speech-power in the brain of the child. By the route of the auditory nerve the names of the common objects which the child repeatedly hears are stored up as 'sound' memories in the auditory centre. As the visual centre grows, the recollections of the shape, form, size, and feel of these objects are registered as visual memories, and by repeated association the name of the object calls up its form or shape, or the perception of its form recalls the memory of its name. Gradually the child is enabled by its motor speech-centre operating upon the lower bulbar centres to pronounce the name of the object. At a later stage it is educated to write the name of the object through the operation of its motor writing-centre.

It is thus evident that the higher speech-mechanism consists of sensory receptive centres and motor emissive centres. The injury to one or other of these will produce sensory aphasia or motor aphasia.

SENSORY APHASIA

is seen in two forms, Auditory Aphasia, or word-deafness; and Visual Aphasia, or word-blindness.

Auditory aphasia may exist without paralysis, and if the lesion causes *complete* destruction of the auditory centre in the cortex (a rare condition) the patient is unable to repeat spoken words—*i.e.* he is word-deaf, and he is unable to write from dictation. He hears ordinary sounds, but his own

language is to him like a foreign tongue which he does not understand. Most people in speaking think more or less of the sounds of the words they are about to utter; hence the motor speech-centre requires the stimulus of the auditory centre for speaking and reading, and when this centre is completely destroyed the patient is unable to understand spoken speech. Owing to the fact that the auditory centre also generally works in association with the visual centre, he is unable properly to comprehend printed speech. For the same reasons he is unable to speak voluntarily or write spontaneously, unless imperfectly, unconsciously making mistakes in spelling and in the use of words. He may be able to copy writing and comprehend music.

Mostly, however, the lesion in cortical word-deafness is only partial, and then the patient can speak fairly, only transposing his words (*paraphasia*) or failing in his nouns, though he may understand what he hears and reads and be able to write from dictation, to repeat words, and read aloud.

In sub-cortical word-deafness the way in for spoken speech is cut off by the destruction of the afferent path from the inferior or primary auditory centre to the auditory speech-centre, this latter remaining intact with its auditory memories unimpaired and in full connection with the other speech-centres. There is then no fault in thought or internal speech, and the case is one of *pure* word-deafness in which the patient is unable to understand or repeat spoken language or write from dictation, though he can thoroughly express himself spontaneously in written or vocal speech and read aloud perfectly. *Amnesia verbalis* is the term used by Bastian to describe functional disability of the auditory speech-centre. The general term, amnesia, is usually loosely employed to designate in its different varieties the conditions characterised or caused by defective memory of words, proper names, or objects.

Visual aphasia, word-blindness, or *alexia*, is the condition in which an educated patient is unable in typical cases to understand written and printed language. His comprehension of spoken words and his power of spontaneous speech is almost normal, but he is unable to write (*agraphia*) and he cannot intelligently copy or use the typewriter, though his central vision may be perfect. His own written or printed language appears to him like the characters of a foreign tongue which he understands not. He may be unable to name objects. As the lesion is generally incomplete, he may be able to recognise some letters or even words, numerals, figures, or music, and he may be partially able to write, but he writes wrong words or places them in a wrong order (*paragraphia*).

In sub-cortical or *pure* word-blindness the path between the half-vision centres and the visual speech-centre is destroyed;

but this latter is intact; hence internal thought and speech are normal. The patient cannot comprehend written or printed speech, and he cannot read aloud, but he can speak and write spontaneously and from dictation, though he cannot intelligently copy. He cannot, moreover, read his own writing, unless by tracing over each letter with a pencil or by means of his finger. There is usually no hemiplegia, but hemianopsia is always present, and sometimes hemianæsthesia on the right side.

Visual and auditory aphasia may exist together, and there may be present mind-blindness or object-blindness (*apraxia*) owing to the partial isolation of the visual centre.

MOTOR APHASIA

This results from destruction of Broca's cortical centre in the third left frontal convolution, and is nearly always associated with right-sided hemiplegia. The patient is unable to speak. With the exception of his being able to say a few words like Yes and No and to ejaculate an oath or some short phrase, he is generally dumb; he cannot repeat vocal speech nor read aloud. There is little, if any, clouding of the intellect, and he is able fairly well to understand written language and to comprehend what is said to him. There is usually agraphia present. Cases differ widely in the degree of their motor-vocal failings, and sometimes a wordless patient can articulate in singing. Where words or short sentences are uttered, it is supposed that their motor speech-memories have been stored up in the inactive corresponding centre in the right cortex. The patient is quite conscious when he makes a mistake in the use of the few words or parrot-cries which he may happen to possess. Where the patient is unable to understand written language (*alexia*) there is also some damage to the visual centre.

Sub-cortical motor aphasia exists when the paths are destroyed which connect Broca's convolution with the lower or bulbar speech-centres. This is what Bastian terms *aphemia*; when the destruction is complete the patient is absolutely dumb. There is no impairment of thought or internal speech whatever; he can even tell the number of syllables in the word which he wishes to utter. He can express himself in writing if his hand is not paralysed, and he perfectly understands both written and spoken speech. This is also called *pure* motor aphasia to distinguish it from the cortical, which is sometimes called *true* motor aphasia. In incomplete cases the speech-symptoms are practically identical with those seen in bulbar paralysis, without the evidence of the paralysis of deglutition, eye-symptoms, &c.

Agraphia is often termed motor-writing aphasia, and, as already stated, there are good grounds for believing that a speech-writing centre exists either as a special centre or as

a part of the hand-and-arm centre, although there are no cases of pure uncomplicated agraphia recorded. The condition is always part of a motor or sensory aphasia, being generally associated with cortical word-blindness, in which case it is complete, or with the sub-cortical form when it is incomplete.

Amimia is the name given to the condition when the patient is unable to understand the ordinary signs of gesture-language, the term *paramimia* being used to designate the state in which he wrongly interprets or employs gesture-language.

Whilst the above-mentioned centres—Broca's speech-centre and the auditory centre—are almost universally accepted as originating the functions implied by their titles, no less an authority than Marie has recently denied that they play any *rôle* in the function of speech. According to the results of the *post-mortem* examinations of fifty aphasic subjects and the clinical study of these in conjunction with the clinical investigation of as many more patients suffering from aphasia, Marie concludes that in every form and all varieties of aphasia the main element is a merely *deficient comprehension of spoken language*. The complicated variations and combinations are brought into harmony by the above conclusion, which implies that all aphasias are merely different degrees of the same disease. Broca's motor aphasia therefore differs only from the sensory aphasia of Wernicke in degree, the patient being unable to speak, whilst in sensory aphasia he is still able to speak more or less imperfectly. As put by Purves Stuart, Marie's view is that Broca's aphasia is simply sensory aphasia minus the power of speech, the essential fact of aphasia of whatever variety being the insufficient comprehension of speech; and when to this latter anarthria is superadded, from a lesion in the neighbourhood of the lenticular nucleus, Broca's aphasia results. Marie maintains that complete or profound aphasia may result when Broca's convolution is intact, and that its destruction is not necessarily accompanied by aphasia.

Diagnosis.—As already mentioned, the difficulty lies in the fact that it is most frequently the case that two or more varieties of aphasia exist together. Owing to the anatomical position and blood-supply of the centres, it is important to remember that the auditory and visual centres are liable to be damaged together, and that the motor speech- and motor writing-centres are also liable to be injured by the same lesion. When the lesion is caused by embolism or thrombosis of the Sylvian or middle cerebral artery, all four centres are damaged and total aphasia results.

It must be kept in mind in investigating any case of complex aphasia that the centres are in their action more or less mutually dependent upon each other, and a lesion which is confined to,

say, the auditory speech-centre will upset the functions of the others. Much also depends upon the peculiarities of the individual, whether, for example, he be a 'visual' or 'auditive' —*i.e.* one who habitually has his visual or auditory speech-centre in a state of greater activity or higher development. Allowance must also be made for the compensatory action of the corresponding centre in the right hemisphere, which in some cases is evidently greater than in others. In diagnosis the fact must be investigated whether the patient is right- or left-handed before the lesion can be located. Marie's test for aphasia is carried out by giving the patient the following series of commands: 'There are on the table three pieces of paper of different sizes: give me the largest piece, crumple up the middle-sized piece and throw it upon the floor, and put the smallest piece in your pocket.' He states that in no case of aphasia, however slight, will the patient be able to follow correctly the above instructions, though he may perform a part of them accurately.

The question of the ability of an aphasic patient to manage his own affairs or to make his will is a serious one in some cases, and no general rule can possibly be laid down. The physician must patiently test the ability of the patient to understand what is said to him and his power of expressing what he wishes accomplished. In one very important case the writer was able to satisfy himself absolutely of the testator's capacity by reading slowly the document to him and occasionally making intentional mistakes which the patient corrected by obvious gestures.

Prognosis.—If the lesion is functional, as in hysteria and migraine, or post-epileptic or toxic, or the result of violent emotion, the prognosis is good. In the organic cases the age of the patient is of great importance. Young subjects easily learn to call into use the centres of the opposite hemisphere, but adults generally fail in the attempt to educate these dormant centres, especially where there is marked hemiplegia, with severe motor aphasia. The nature of the organic cause has an important bearing; thus arterial degeneration is almost certain to be progressive, whilst embolism gives a better outlook. Partial recovery is, however, possible in the worst cases if the affection is recent, especially in cases of word-deafness; and the prognosis is better in the sub-cortical lesions where the intact centre is merely cut off.

Treatment.—The removal of the cause in the functional cases by the elimination of poisons, reduction of blood-pressure, and the improvement of the general nutrition, becomes the obvious object of the treatment. For the improvement of the aphasic condition itself, the hope of success must mainly rest upon attempts to educate the speech-centres of the sound hemisphere, as if the patient were a child. He should be

patiently taught his letters and gradually trained to spell and afterwards to write by using his left hand. In motor aphasia he should be taught to continually practise the simplest speech-movements, beginning with words and gradually proceeding to their simplest combinations.

APOPLEXY. (See CEREBRAL HÆMORRHAGE.)

APPENDICITIS

Inflammation of the vermiform appendix and of the peritoneum in its immediate vicinity was formerly called Perityphlitis and Typhlitis. Though these names are still retained by a few writers, by common consent the appendix is regarded in nearly every case as the origin or starting-point of the affection.

Etiology and Pathology.—The appendix varies considerably in its size and condition. Averaging three and a-half inches in length, it lies mostly behind the end of the ileum or the cæcum with its tip pointing in the direction of the spleen. Its diameter is about a quarter of an inch; its walls are thick and very rich in lymphoid tissue. It has no use or function, being merely the obsolete representative of the conical cæcum still existing in apes.

Inflammation of the appendix is most common in men (nearly four to one), and about half the cases occur before twenty.

A catarrhal inflammation starting in the cæcum is the cause of the hard concretions so frequently found in the appendix and so often mistaken for foreign bodies. The presence of these may lead to obstruction, stricture, ulceration, gangrene, or perforation, though such results may ensue from the inflammatory action alone in the absence of concretions. Foreign bodies, as seeds, bristles, &c., are sometimes, but not often found ; intestinal parasites have been demonstrated as the causal factor in some cases.

In chronic cases the lumen of the appendix may be found narrowed or almost obliterated from old standing inflammation. This seriously interferes with the drainage of the organ, as is especially seen in the recurring cases, and may at any moment become the cause of rapidly severe or fatal ulceration, perforation, or gangrene from the supervention of an acute septic process. Other causes, as torsion, kinking, injury, tubercular and typhoid ulceration, &c., may set up the attack.

The most important element in an attack of appendicitis is the peritonitis. This is caused by the local invasion of the membrane in probably every case by virulent *Bacillus coli*.

streptococci, and occasionally by *Staphylococcus pyogenes aureus*, which pass through the walls of the appendix, facilitated by previous inflammation or ulceration, or by the injuries produced by masses of decomposing or indigestible food in the cæcum. The *rôle* played by the above microbes and even by the *pneumococcus* is now universally accepted as the main element in the etiology of appendicitis, the presence of foreign bodies being regarded as of little importance. The peritonitis is fortunately in the great majority of cases localised; and even when the process ends in suppuration, the abscess is confined to the right iliac fossa, being shut off from the general cavity of the peritoneum by adhesions between the neighbouring coils of bowel. The abscess-cavity is situated deeply, and may be found to contain ill-smelling pus and gas; the appendix may be found perforated, gangrenous, or completely detached; this condition is liable to occur in the most dangerously acute cases, where pus may form as early as the third day. The abscess, if left to itself, may burst into the neighbouring bowel, bladder, or vagina, or it may discharge through the skin of the abdominal wall, or pass below Poupart's ligament or even ascend into the pleura.

Symptoms.—These vary considerably, the most constant and prominent being severe sudden *pain*. This is generally referred to the region of the umbilicus, but in from twelve to twenty-four hours it becomes localised over the appendix. Almost from the first, marked *tenderness* can be elicited over McBurney's point. This is the spot where the outer edge of the right rectus muscle intersects a line drawn between the anterior superior iliac spine and the umbilicus.

Nausea is very common and *vomiting* is often present; it generally subsides early, unless in severe cases, when it may be constant.

Constipation is the rule, though sometimes the bowels continue to act regularly.

Fever is generally present, except in some of the perforative cases with rapidly grave septic symptoms, when the temperature may be even subnormal though the patient be suffering from well-marked general peritonitis and collapse. The temperature often reaches its height during the second or third day, after which it gradually declines in those cases which do not go on to suppuration, and the normal is reached by the end of a week. It is, however, very variable, and is sometimes high in the most favourable cases. A prolonged high temperature, or a marked elevation after the normal has been reached, is highly suggestive of suppuration.

A *tumour* forms beneath the tender spot, though at first there may be only evidence of increased resistance under deep pressure. As Treves points out, the magnitude of the

swelling is not in proportion to the gravity of the case, some of the most fatal cases showing only slight swelling. Dulness over the tumour often cannot be elicited owing to coils of bowel entangled in it. The swelling generally disappears gradually, but thickening may be felt for weeks after recovery.

Suppuration occurs in some rare cases as early as the third or fourth day; it is, however, generally later, and it must be remembered that the majority of cases get well without abscess-formation.

When pus forms, all the symptoms deepen: pain, fever, swelling, tenderness, abdominal distension, rapidity of pulse, and dryness of the tongue give evidence of the serious nature of the patient's condition. The pain extends down the thigh or into the testicle, and the limb is drawn up; sweating, rigors, vomiting, and signs of general peritonitis may supervene. Fluctuation may be detected, and sometimes gas accumulates in the tumour; and unless the abscess be speedily incised, rupture into the peritoneal cavity may occur.

In those cases where perforation or sloughing of the appendix occurs very early, the attack from the first is of the fulminating type, and the symptoms are those of an acute general perforative peritonitis caused by the escape of virulent micro-organisms into the peritoneal cavity.

Relapsing or Recurrent Appendicitis.—This is the name given to a rather common condition in which the patient, after complete recovery, experiences a number of distinct attacks at irregular intervals. The severity and number of the separate attacks vary greatly, and in the intervals the patient is well and free from symptoms. This type is not found in those patients where suppuration has occurred during an attack of appendicitis. In the majority of cases the recurrences follow indiscretion in food, exposure to cold, or trauma.

Diagnosis.—As a rule this is easy, especially in males, where the chances are at least fifty to one that any peritoneal inflammation is of appendicular origin.

Given, then, the symptoms of peritonitis, the *tubercular* variety is easily excluded by attention to the history of the case, which is slow, giving evidence of alternating constipation and diarrhœa, with wasting. In those cases where a tubercular ulcer perforates in the region of the cæcum the diagnosis is difficult, and only careful sifting of the previous symptoms will enable the physician to come to a correct conclusion. The same remarks apply to the perforation of *cancerous ulcers.*

Typhoid fever may lead to a localised peritonitis in the right iliac fossa which has been mistaken for appendicitis, and should the ulcer perforate, only the other signs of the disease

will enable a diagnosis to be made. Widal's reaction is of much value in these doubtful rare cases, and enlargement of the spleen is nearly always present.

Gastric and *duodenal ulcers* which have perforated may simulate appendicular peritonitis, but the previous history even in latent cases generally reveals the fact of black motions having been noticed at some antecedent period.

Pelvic peritonitis in the female must be excluded by the absence of the history and signs of pyosalpinx, of pelvic hæmatocele, and of ovarian disease, and by the absence of rigidity of the abdominal muscles.

Intestinal obstruction and *intussusception* when peritonitis has supervened may closely resemble appendicitis; in the former the pain and collapse are more marked, and the distension of the abdomen and vomiting are severe. In intussusception the tenesmus and bloody stools with colicky pains, and a movable and not very tender tumour in the iliac region, and the early age of the patient, are characteristic. In both cases the temperature may be subnormal. (See under 'Intestinal Obstruction'—Diagnosis.)

Distended and inflamed gall-bladder, when the peritoneum has become involved, can be differentiated by defining the lower border of the tumour, which is more or less movable, and the history assists in clearing up the diagnosis.

Several conditions in which there is no suspicion of any peritoneal inflammation may simulate appendicitis—

Renal colic is differentiated by the absence of fever, the severity and constancy of the pain, which radiates downwards into the testicle and bladder, and by the presence sometimes of irritable bladder and bloody urine and tenderness over the kidney near to the spine behind.

Hepatic colic, with its sudden severe pain and normal temperature, is often ushered in with a rigor and smart vomiting; tenderness is marked under the costal arch, and the pain beginning in the gastric region radiates to the shoulder and jaundice soon supervenes.

Mucous colitis is slower in its advent; the temperature is not elevated; the pains are distinctly colicky and not localised; and the motions contain the characteristic mucous shreds.

Neuralgia of the abdominal muscles, *hysteria*, and *enteralgia*, and the pain produced by displacement of a *floating kidney* are as a rule easily excluded by the continued absence of fever and the localisation of areæ of tenderness.

When the physician sees the case for the first time after suppuration has occurred, the question of a *psoas abscess* may arise, and this can in many cases only be differentiated by a very close scrutiny of the history of the case—the onset in appendicitis being sudden, and the deformity of the spine, with rigidity, being characteristic of psoas abscess.

☛ In all doubtful cases a count of the blood-cells should be made, as the researches of Curschmann have proved that leucocytosis occurs in the majority of all forms of appendicitis from the commencement, though several authorities deny the frequency of this sign. The number of leucocytes may reach 20,000 in a cubic millimetre of blood upon the second or third day in a case where no suppuration occurs. The number rapidly falls to half, or less than this amount (7,000 to 8,000 represent about the normal). Where the leucocytosis remains high, or exceeds upon *repeated* examinations the above maximum, it may be safely inferred that suppuration has taken place, and the blood-count under these circumstances is of more value than the evidence of the temperature, pulse, and other physical signs. Thirty-five to fifty thousand leucocytes have been found in suppurating cases, when the temperature showed a return to the normal; hence the importance of repeated estimations of the number of leucocytes cannot easily be exaggerated, both as regards diagnosis, prognosis, and treatment. In some fulminating cases, where general septic peritonitis of a virulent type is present, the number of leucocytes may not be markedly increased owing to the natural resistance of the body being markedly reduced; hence, as pointed out by Gazin and Gros, under these circumstances the absence of leucocytosis must be looked upon as an extremely serious prognostic sign. It should also be remembered that leucocytosis may be entirely absent in cases where an encysted abscess persists after the disappearance of the acute symptoms.

Prognosis.—This is very favourable in the non-suppurative cases, few, if any, of which ever end fatally. Treves puts the mortality of all cases, including the most severe and the most trifling, at about 5 per cent. The prognosis is always serious in the suppurative cases; but it is largely influenced by early operation, statistics showing a mortality of about 15–20 per cent. after operation. Treves calculates that when an abscess occurs the risk to life is at once raised to 30 per cent. This latter figure will probably be doubled where no operation is the rule, and it will probably be halved when early incision is resorted to. The importance of a blood-count in weighing the question of prognosis has already been discussed under Diagnosis.

The prognosis in relapsing cases, when the operation of removal of the appendix is performed during the interval after the attack, is always good, the mortality being less than 1 per cent.

Treatment.—This is discussed at length in the author's 'Dictionary of Treatment' (4th ed.), pp. 696–699, and only the general principles need be here referred to. During the attack the remedies to be employed are those indicated

in peritoneal inflammation—rest in bed, a moderate dose of morphia to relieve pain, an enema to unload the bowel, or, if seen in the very early stage, one small dose of castor oil or of calomel ; severe or continuous purging and large doses of narcotics are to be condemned. The application of warm poultices, or of the ice-bag, and at a later stage of leeches, is indicated. Small quantities of iced milk by the mouth should be given frequently, and when vomiting is severe rectal feeding must be resorted to. In the majority of cases these measures are successful ; but when by the end of the fourth or fifth day the condition of the tumour, the temperature and pulse remaining high, affords evidence of suppuration, a free incision is imperative. There is great divergence of opinion and of practice regarding the period at which operative interference is necessary. American surgeons insist upon operation as early as the third day—*i.e.* forty-eight hours after the onset. The English practice is, unless in very exceptional cases, to postpone operative measures till the fifth day or later. The question really resolves itself into the simple one of the evidence of the presence of pus. It must be laid down, as a rule, that when pus has formed the abscess should be incised without delay ; but the pulse, temperature, and local signs are often misleading, especially when morphia has been freely given, and the surgeon who makes a rule of opening the abdomen forty-eight hours after the onset of symptoms of appendicular trouble will certainly submit many patients to the risk of operation who would recover if treated by the expectant plan.

A free incision should be made through the abdominal parietes over the most prominent part of the tumour, dividing the transversalis fascia and peritoneum ; if the appendix comes into view it should be removed. A free evacuation of the abscess-cavity and its thorough drainage are generally all that is required. Most authorities condemn the use of the exploring needle before operation. Where the pus has already made its way into the peritoneal cavity, this must be thoroughly cleansed and the various routine measures indicated in general perforative peritonitis should be resorted to.

The after-treatment will consist in an early evacuation of the bowel ; if there be much distension, a small dose of morphia and the cautious administration of liquid nourishment in limited amounts by the mouth are indicated.

The treatment of relapsing appendicitis in the intervals between the attacks resolves itself into a careful regulation of the bowels and of diet, and the following remarks apply to all cases of the disease in which recovery has followed an attack, especially where suppuration has not occurred, as such are always liable to relapses. The diet should consist of most easily digested materials, only such as leave the

least indigestible residue being selected. Nuts, seeds, rinds of apples, gooseberry and grape skins, &c., must be forbidden, as also the stringy, fibrous, and cartilaginous portions of meat. Meals must be taken at regular intervals and slowly and most thoroughly masticated ; to ensure this, the condition of the teeth must be carefully attended to. The bolting hurriedly of solid food often determines an attack.

The bowels must be daily evacuated, and upon the first warning of an attack a full dose of castor oil should be administered. Exposure to cold and wettings, and severe exercise, must be avoided.

There is much difficulty in deciding, in a given case of relapsing appendicitis, whether the patient should be submitted to the operation for removal of the offending organ. In deciding, it must be remembered that the mortality of the operation, if performed in a quiescent period, is under 1 per cent. ; hence, if the attacks are numerous and severe, and especially if they show a tendency to become more dangerous, or if they interfere with the discharge of the patient's life-duties, the appendix should be removed without waiting for the risk of another attack. Many surgeons insist upon the necessity of the removal of the appendix after the second attack.

ARTERIO-CAPILLARY FIBROSIS

This disease, which is characterised by evidences of a chronic universally distributed thickening of the arterial walls, was so called by Gull and Sutton, and is also known as Arterio-sclerosis, *Atheroma* or *Endarteritis deformans* being a successive stage of the pathological process. The terms unfortunately lead to confusion, because this latter condition may exist in a form so apparently different from arterio-sclerosis that it is by some regarded as a different pathological entity, though these authorities recognise that both conditions often exist together.

Pathology and Etiology.—As the name implies, in the typical diffuse condition there is a general fibroid degenerative action beginning in the walls of the small vessels. The internal coat shows fibrous overgrowth which is believed to be secondary or compensatory to a weakness of the middle (muscular) coat. There is a general thickening of the arterial walls, and a hyaline change is well seen in the capillaries of the kidney and brain, but the *entire vascular system* is progressively involved. In some subjects the fibroid growth affects the vessels in the kidney to a greater degree than those in other regions, and the result is the establishment of the so-called fibroid or contracted kidney of Bright's disease. In others the coronary arteries suffer most and cardiac degeneration

with angina becomes established ; in others brain softening or apoplexy follows.

In all cases there is a central pathological condition induced owing to the obstruction of the flow through the capillaries and small vessels. The blood-pressure rises permanently, and to overcome this the cardiac muscle must hypertrophy greatly to carry on the circulation efficiently. The increased vascular tension tells chiefly upon the large arteries. These, from the aorta backwards, become the seat of atheroma or endarteritis deformans, which may be of the diffuse or nodose variety ; gelatinous patches of degeneration begin under the endothelium : these become dense like cartilage and may calcify in hard plates, and the so-called atheromatous abscesses may be present ; with these, hyaline changes in the muscular coat are observed, and the elastic fibres may rupture from the strain so that aneurisms form. In some cases, especially in the cerebral vessels, a periarteritis is present alone, but nearly always in association with the endarteritis ; this is seen best in the syphilitic form of the disease, and may end in the formation of nodular or caseating gummata. Changes are also not uncommon in the veins : these may be found the seat of considerable thickening, especially in their inner coats ; rarely are atheromatous deposits found.

The causes of this universal and progressive vascular change are numerous. It may be the result of the usual senile change always found in the aged, and which shows itself in some individuals as early as late middle life. The following are potent factors in determining its onset—viz. *syphilis* ; the poisons of *gout*, *lead*, *alcohol*, and *phosphorus* ; continuous severe *muscular strain* ; continual *over-feeding*, and some forms of *desquamative nephritis*, and *hepatic disorders* which diminish the power of the liver to destroy various ptomaines and leucomaines. Great difficulty exists in demonstrating the manner in which these agents act. Some authorities affirm that the *materies morbi* circulating in the small vessels produces contraction of their coats which leads to increased general arterial tension, and that this is the factor which produces the arterial thickening, which is thus of a compensatory nature at first. Others maintain that the starting-point of the disease is the increased viscosity of the blood which is produced by the toxins circulating in it ; this gives rise to increased blood-pressure. The sharpest differences of opinion have concentrated around the renal form of the disease—one view being that the change in the renal capillaries is the cause of the general arterio-sclerosis, the other view being that the general condition causes the renal lesion. The safest hypothesis is to regard the renal affection as but a part of a universal and progressive vascular change, probably different from that found in the typical granular kidney of Bright's disease.

Allbutt has long maintained that there is no direct relation between the arterial tree and blood-pressure, and that often in arterio-sclerosis the blood-pressure is constantly low; this is probably always the case in the senile form. He lays stress on the viscosity of the blood as the potent cause, the increased arterial pressure in its turn causing sclerosis of the vessels. The disease shows itself in men after forty and in women generally a decade later.

Symptoms.—The symptoms and signs are those mainly of increased arterial tension, with a high-pressure pulse and markedly accentuated second sound of the heart, followed by steady hypertrophy of the left, and to some extent of the right ventricle. The sphygmogram shows the absence of the percussion-wave, with a short sloping upstroke and a slow downstroke, with little dicrotism. The thickened and overfilled radial may with difficulty be obliterated by the pressure of the finger, and then the degenerate rigidity of the vascular walls may be easily recognised below the obliterated point. The temporal arteries will be seen to be tortuous, and to the finger their walls are rigid. The urinary secretion is generally markedly increased, and if very careful examinations be made at regular intervals, at some time or other transient but slight albuminuria is certain to be found; usually it is absent till the later stage supervenes, when it is pretty generally present, but always in small amount.

In this condition the patient may remain without symptoms of any kind for long periods—often for many years, when one of several events may suddenly occur:

1. Cerebral hæmorrhage is very likely to supervene. It may be preceded by any of the transient hemiplegic or monoplegic attacks, vertigo, aphasia, or unconsciousness, seen in syphilitic cerebral arteritis.

2. Anginal and syncopal attacks from coronary disease, sometimes preceded by very slow pulse, are not uncommon. Sudden dilatation of the heart, with breathlessness, severe palpitation, and even alarming dyspnœa, may herald the advent of a rapid failure of compensation under some trifling muscular exertion. The sudden increase in the capacity of the left ventricle prevents the perfect approximation of the mitral segments, and regurgitation with the formation of a loud apical systolic murmur is audible; and should prompt treatment prolong life, dropsy, pulmonary congestion, and other symptoms develop. Persistent breathlessness, with a loud second sound which may be reduplicated, and pulmonary emphysema, may be the first symptoms to attract attention; and, as Allbutt forcibly puts it, the fall of pressure (not the result of treatment) has a serious import, and when the patient begins to be short of breath he has entered upon the last stage of his pilgrimage.

3. Renal asthma or uræmic convulsions, generally without any anasarca, and with a copious pale urine of low s. g. and little or no albumin. Renal inadequacy, so-called, has probably no direct relationship to arterio-sclerosis.

Diagnosis.—This is easy if the physician grasps the idea of a general vascular degeneration of a progressive nature causing a constantly high blood-pressure in all the arteries within sight and reach, which become thickened, rigid, and tortuous, eventually leading to hypertrophic changes in the left ventricle, accompanied by a highly accentuated aortic second sound.

Prognosis.—This is always grave, owing to the dangers of cerebral apoplexy and cardiac degeneration; but much may be done to arrest for the time the progress of the disease. Those who consider that the high arterial tension precedes the vascular thickening believe that the disease is curable at an early stage. Prognosis is very bad in those cases where it is not possible to remove the cause of the sclerosis, as in alcoholics and persons subjected to prolonged muscular strain. The prognosis is always grave in those breathless cases where a previously constantly high pressure has given way to a constantly low one.

Treatment.—This will consist in the use of remedial agents which tend to counteract the causes at work in a given case; thus lead, alcohol, syphilis, and gout afford clear indications. Muscular fatigue and prolonged muscular strain must be avoided, and, in a word, everything which increases the already high pressure of modern life, with its intense business worries and stress, must be given up. The periods of work, rest, and exercise must be carefully mapped out and adhered to. Diet should be spare, light, and unstimulating; a change to vegetarianism often gives excellent results; in every case butcher meat and strong animal soups should be avoided, but fish may be freely used; alcohol in all forms, save in that of a limited amount of light claret, is objectionable. Of all the factors for good, none are so potent as a restricted diet and graduated muscular exercise. For the diseased condition itself, the only benefit from drugs will be found in the use of iodides in full doses, for long periods at a time. Arsenic may be combined with iodides, but the dose should be small, and the drug only given for a few weeks without a pause. Some recent writers, including Morison, speak strongly of mild mercurial courses in the form of Guy's or Baly's pill as possessing an almost specific action.

Vaso-dilators like nitroglycerin are of course valuable, but their effects are so evanescent that to get any real advantage they must be given *very frequently* in small doses in a totally different way from that usually recommended, so that the

arterial pressure be not permitted to rise until another small dose be administered. For this purpose a small tabloid containing $\frac{1}{100}$ gr. nitroglycerin may be swallowed every hour whilst awake; but few patients will be found who can be depended upon to keep up a course of this kind for more than a few weeks at a time. Such a course may be imperative in the presence of a threatening cerebral hæmorrhage, uræmic attack, or anginal condition. The complications that arise are to be treated upon the lines laid down for the disease of the affected organ. Great benefit is obtainable from the constant use of saline purgatives, as constipation must be guarded against; hydropathy, or a residence for a portion of each summer at a saline spring, may be recommended.

ARTERITIS

The acute form of inflammation of arteries is generally a local one set up by some septic or suppurative process in the immediate neighbourhood of the vessel, or by a septic embolus brought from a distance, or by some virus circulating in the blood which causes injury to the inner coat or which is brought to a spot in the circulation where injury has already prepared a vulnerable spot for it. This latter condition is seen in the arteritis following the shedding of calcareous particles from the cardiac valves, and, according to Osler, affords an explanation of the cerebral hæmorrhages occurring in youth. The local forms of arteritis tend to eventuate in hæmorrhage or aneurism. General acute arteritis is always the result of septic infection. It is seen in ulcerative endocarditis, influenza, acute rheumatism, and other fevers; the writer has seen it in a typical form following small-pox.

The symptoms vary, and the affection can often be only recognised by the local results which follow the obstruction of the vessel, and these are generally the symptoms indicative of gangrene—*i.e.* pain and numbness, followed by complete anæsthesia and œdema.

Syphilitic arteritis occurs during the early years of syphilis, and constitutes the fundamental element in the pathology of syphilis. According to Marshall, it may attack any artery in the body, but it chiefly selects the cerebral vessels about the base of the brain and Sylvian fissure. All the coats are involved, and the ultimate result may be obliteration of the lumen of the vessel or an aneurism. As mentioned elsewhere, this condition is a powerful factor in the formation of aortic aneurism.

The obliterative tendency, in the case of the brain-vessels, produces symptoms which are characteristic of brain-syphilis; mental functions are depressed and transient, paralyses—

monoplegic or hemiplegic—alternate with aphasia, syncopal attacks, impairments of vision, and sensory disturbances. The transitory nature of these seizures is explained by the slow nature of the occlusive process, which permits of collateral establishment. At a later stage, when the collaterals become blocked, the symptoms are permanent. Mott rightly insists upon the vital importance of energetic treatment in the early stages by inunctions combined with large doses of iodides.

According to Weichselbaum, syphilis is the cause of the strange and rare form of *periarteritis nodosa*, which rapidly eventuates in the formation of nodular swellings in the small arteries of the body. The subject of arteritis is considered in detail in the article on 'Syphilis.'

Tubercular arteritis is seen in tubercular meningitis and phthisis; in the latter case, the invasion of the coats of the pulmonary arterial branches by the bacilli leads to occlusions or small aneurisms which are often the cause of hæmorrhages.

ARTHRITIS DEFORMANS

This is a chronic progressive affection of the joints, probably caused by microbic infection, resulting in specific destructive changes in the cartilage and synovial membrane, with hypertrophic changes in the articular margins of the bones and muscular atrophy.

The disease is known under various other names, as Rheumatoid Arthritis, Osteo-arthritis, Chronic Rheumatic Arthritis, and Rheumatic Gout. The two latter terms should be discarded, as the affection is distinct from the protean forms of gout and articular rheumatism.

Etiology and Pathology.—Whether its pathogeny be that of an infective process, a senile degeneration, or of nervous origin as in the joint-affection of tabes, authorities widely differ; but it cannot be denied that cold and damp act as powerful exciting or predisposing causes, especially when combined with any condition causing general debility, as prolonged lactation, menorrhagia, insomnia, grief or prolonged worry, under-feeding, anæmia, &c.

It is more common in females, beginning about middle life, though it is found in children not infrequently, and often the senile changes of old age seem to merge into it. Occasionally it follows severe influenza and repeated attacks of acute articular rheumatism. In Ireland, where it is very common, the writer has often traced a history of heredity, of phthisis, and sometimes of cancer. These, however, are probably only predisposing agencies which leave the patient open to the attack of the various infecting organisms whose existence

in the joints has been observed by several. The association of tubercle with the disease will be referred to later on.

Morbid Anatomy.—The changes in the joint are characteristic: the cartilage softens, splits into fibrillæ, and gradually becomes absorbed, permitting the bones to come into direct contact; their articular surfaces become polished, and the eburnation causes the superficial layer of bone to appear like porcelain. Osteophytes develop round the articular margins, and these may cause locking or false anchylosis; the ligaments and capsule thicken whilst the tendons waste and the muscles atrophy, the nerves showing signs of chronic neuritis. The ultimate result is great deformity. These changes in the joints of the fingers are recognised as Heberden's nodes, which are little knobs caused by osteophytic outgrowths at the sides of the bases of the distal phalanges. In the hip-joint the cavity may become so deepened that the wasted head of the femur slips out of it and forms a false joint, the ligamentum teres having disappeared; the disease at the metacarpo-phalangeal joints causes great deformity by producing deflection of the fingers towards the ulnar side. Every joint in the body may become affected, the maxillary and vertebral not infrequently. The characteristic joint-changes have been observed by the writer in the partially fossilised bones of the extinct Irish elk (*Megaceros Hibernicus*). Whilst these changes are present in the joints, the shafts of the bones remain normal, or sometimes exhibit hypertrophic changes, the osseous tissue appearing often to be unusually dense. Cardiac lesions are occasionally found, as in rheumatic cases, but the percentage is so small that their presence must be regarded as purely accidental and possesses no pathological signification.

Symptoms.—These are variable at the commencement, and at least two well-marked types of the affection are recognised.

The *acute* form of the disease constitutes one of the most serious of maladies. It begins with joint-pains in the hands and feet, with moderate increase of temperature in young subjects, mostly girls or young married women, especially during *prolonged lactation which has been overlapped by pregnancy*. There is sometimes considerable sweating without fever, the perspiration being very different from that of acute rheumatism; the joints become markedly tender and swollen, and the tendency is for the affection of the joints to spread towards the centre from the periphery. The characteristic deformity soon appears in the finger-joints, and in a few months all the other joints may give evidence of serious change, causing permanent crippling of the most distressing nature. The clinical picture presented by these acute cases is one of the most serious and dreadful, the patient being rendered absolutely helpless and bed-ridden for life, often unable to move

any joint in the body save the jaw or vertebræ. Some of these cases, however, where the predisposing debility is of a removable kind, cease to progress for a variable time, and amelioration of the symptoms may take place without apparent change in the deformed joints. Cases occasionally occur in which the onset is as rapid as in the most grave type, but after a few weeks the disease passes imperceptibly into the common slowly progressive and chronic form.

In the *chronic* form of the disease, which is by far the most frequently to be met with, the affection is very insidious, and it should always be borne in mind that the earlier characteristic joint-changes have been found in cases where the subject during life had never complained of any symptom of the affection. The earliest symptom is a pain and stiffness in one or more joints following active exercise; in many instances these sensations are not experienced till the patient attempts to get up after resting, and this constitutes one of the most characteristic features of the disease in its early stage, when involving the hip, knee, or ankle. The pain is, moreover, markedly aggravated by changes in the weather, and it seems strange that this is very noticeable when a large deeply seated joint like the hip is involved. The patient becomes highly susceptible to cold, and generally keeps the affected joints, and often the entire body, buried in superfluous layers of clothing, though sometimes heat and sometimes cold aggravates. In the monarticular form of the disease, which is the variety most common in men, it has been noticed that the disease often begins in the joints nearest to the trunk. In the polyarticular form, which is most common in women, the fingers first suffer, and the tendency of the disease then is to involve gradually the joints in a centripetal manner; but as a general rule it may be said that the joints which suffer most are those most subjected to wear and tear, and in these especially large amounts of synovial fluid may accumulate and bursal swellings occur in their vicinity. Pain varies much in character and intensity, and it is always greatly aggravated by movement even before deformity becomes marked. The advent of early neuritis may entirely alter the clinical picture and mislead the physician, as the pain may radiate away from the joint and entirely overshadow the local symptoms, where, in the case of the hip, it may be regarded as a true sciatica, and in the shoulder as neuralgia. Wasting generally is out of proportion to the enforced rest of the joint, and often seems to be an accompaniment of the neuritis; weakness of the muscles of the affected limb is always present. A peculiar fine creaking or crepitation may be felt and heard at an earlier stage than is generally recognised, and is to be differentiated from the coarser phenomenon experienced on the movement of joints in which osteal outgrowths have formed at a later stage

of the malady. Local and general sweating is often noticeable, and Spender lays stress upon the presence of a constantly quick pulse and of patches of bronzing or increased pigmentation of the skin resembling round freckles on various parts of the body, notably on the fore-arms and face, and generally associated with great coldness of the hands. A marked glossiness of the skin is common. In some cases, in addition to the deformity produced by the changes in the joints, there is added muscular contracture, which completely cripples the patient and compels him to spend the remainder of his life in bed.

In young children, cases are met with apparently identical with the acute progressive polyarticular form sometimes seen in adults. A peculiar arthritic affection has been described by Still, under the name of Arthritis Deformans, occurring in children before the period of second dentition; it is, however, a totally distinct disease. There is, in addition to the progressive enlargement of the joints, always an increase in the size of the spleen and of the lymphatic glands. The onset may be sudden, with fever, or insidious when only a few joints are at first involved. Soon knees, wrists, elbows, feet, and hands are symmetrically attacked, the enlargement involving the soft tissues and never ending in the characteristic osteophytic growths peculiar to the disease in the adult. The lymphatic enlargement is chiefly confined to the glands in the neighbourhood of the affected joints, but it may become general. The progress may be slow, but always ends in great deformity, chiefly owing to muscular contracture, and the stunted little victim becomes a cripple for life.

Edsall and Lavenson have lately investigated a number of these cases, and by the marked reactions which followed the injection of tuberculin they were led to accept Poncet's theory of the intimate association of tuberculosis and rheumatoid arthritis. In some instances of the ordinary type of the disease in adults, the injection of the synovial fluid into guinea-pigs was found to produce markedly tubercular lesions.

Diagnosis.—*Rheumatism* is generally easily excluded, by the absence of marked fever, but there is considerable difficulty in the rare form of arthritis deformans, which comes on acutely and affects rapidly all the joints of the hands and feet in subjects about the age of thirty. The physician must watch the progress of the case for some time before a diagnosis is possible; the presence of a tuberculous history with concurrent prolonged lactation, menorrhagia, ovarian disease, or leucorrhœa, and much debility, especially if the arthritic pains are not markedly relieved by full doses of salicylates, should arouse a strong suspicion before the characteristic nodes and joint-deformities appear. The presence of a very

quick pulse where there is little or no fever, and the appearance of Spender's pigmentation on the fore-arms or melasma on the face, are valuable diagnostic points, especially when accompanied by marked local muscular wasting and cold sweating of the hands and feet. The chronic form is generally easily differentiated from chronic articular rheumatism by the obvious joint-deformity, especially in the absence of a clear history of antecedent attacks of acute rheumatism and of heart-symptoms.

Gout is easily excluded by the absence of urate-of-soda concretions in the skin around the joints or in the helix of the ear, by the negative result in testing the blood for uric acid, and by the history of the onset, which in *first* attacks of gout consists always of an assault on some one small joint.

Synovitis, following injury, and *tubercular* disease of a joint may simulate arthritis deformans, but the history of the case generally suffices to establish the diagnosis ; and even of more importance is the examination of the other joints, some of which certainly will give evidence of A. deformans if this affection is present. *Gonorrhœal synovitis* is recognised by the history of the specific infection and the detection of the gonococcus which is often present in the effusion. The joints affected are often the sacro-iliac, sterno-clavicular, intervertebral, and temporo-maxillary.

When A. deformans attacks the joint of the jaw and the vertebral joints, notably the cervical, the symptoms are fairly characteristic, but neuritis or neuralgic conditions may overshadow the purely local joint-mischief. In these cases the diagnosis is often rendered obvious by the great stiffness, rigidity, or immovability of the joint.

Charcot's joints, seen sometimes in ataxia, may in rare cases resemble the monarticular cases of A. deformans. Too much stress has been laid upon this resemblance because of the theory that A. deformans is a neurosis. The joint-changes are certainly allied, if not apparently identical, both as regards erosion of cartilages and change in the articular surfaces ; but the destruction of the joints which occurs sometimes in ataxia comes on with startling suddenness and progresses with lightning rapidity, the first symptom being an extensive painless effusion.

Progressive muscular atrophy may resemble those cases where marked weakness and wasting appear early in the hand and fore-arm muscles, but the absence of pain, nodes, local sweating, and other joint-changes, generally settles the diagnosis.

Hypertrophic pulmonary osteo-arthropathy is often mistaken for rheumatoid arthritis by those not familiar with the existence of such a disease ; the chief point in the differential

diagnosis is the causation of this interesting malady as implied in its name, being always associated with pulmonary lesions of a chronic type, as phthisis, chronic bronchitis, pleuritis, or empyema. The enlargement is mainly confined to the bones of the hands and feet, the fingers and toes being longer and thicker than normal, the ungual phalanges always clubbed, and the nails much striated and curved. There is commonly some swelling of the wrists and ankles.

Osteitis deformans is easily differentiated by the peculiar enlargement of the head, which does not involve the face; though the arm- and leg-bones are enlarged and distorted, the bones of the hands and feet escape, or are but slightly involved, and the deformity does not obviously involve the joints.

Prognosis.—As a rule, the disease is progressive, and in the acute polyarticular form the prognosis is most grave as regards the crippling of the patient for life; but even here the affection may halt in its maiming progress for shorter or longer periods. The disease has no tendency to shorten life. If we regard the appearance of Heberden's nodes as characteristic of A. deformans, it must be acknowledged that the diseased process may become arrested permanently, for some patients do not suffer further trouble. Even in severe cases of the monarticular form, as in the hip, knee, or shoulder, when the joint-disorganisation has become complete, the pain and distress may stop entirely and the patient may only suffer from the mechanical difficulty caused by the stiffness of the joint. Some measure of recovery for long periods has been observed to take place in the knee even when the effusion has been so great as to constitute the typical condition known as hydrops articuli; the general tendency, however, is for the joint-trouble to become symmetrical and to extend to the smaller joints, especially to any which have become the seat of strain or injury.

Treatment.—As a rule, this is most unsatisfactory. Nevertheless, much may be done to relieve the patient's distress and to minimise the evils of his deformity, and in not a few cases to retard the progress of his malady ('Dictionary of Treatment,' pp. 851–856). As soon as the disease is recognised, removal to a dry uniformly warm climate is essential if improvement is to be expected; and this is especially imperative in regard to cases originating in the cold and damp climatic conditions prevalent in Ireland, where the disease is very common. The winters of Algiers and Las Palmas are suitable, and summer may be spent at any inland English health-resort, as it is generally found that the pains are aggravated by proximity to the sea. The locality should be selected with the view of obtaining the best thermal or hydropathic arrangements, such as exist at Bath, Harrogate, Aix-les-Bains, Wiesbaden, &c.; and in this matter the local joint-

treatment by various forms of massage, douches, &c., is of more importance than the nature of the medicinal water or spa.

Diet is of much importance and should be most liberal. The worst cases met with are those in which the patient has been led to adopt an anti-gout diet which seems to hasten the march of the disease: plenty of meat and a moderate amount of stimulant, unless when specially contra-indicated, should be taken. The importance of attention to the nature and amount of clothing worn is only second to the climatic question. Light flannel underclothing with chamois interlinings over the affected joints is the best covering. The writer is satisfied that over-clothing is worse than too little clothing, which in itself is a very serious mistake. He has been sometimes tempted to believe that the disease may possibly be caused by wearing too much clothes, after remarking the rapid progress of the joint-mischief in patients who wear so much clothing as to be in a state of constant perspiration. Monarticular cases wearing excessive clothing are almost certain to have the hands soon affected, for it would seem that *to keep most of the joints too well covered is to reduce the resistance of the exposed ones.*

It is needless to remark that every departure from the laws of health is to be corrected, as regards work, rest, sleep, regularity of meals, ventilation, prolonged excitement, menstrual disorders, &c. Drugs are of very secondary importance: no known substance exercises any specific influence over the disease. Arsenic in full doses combined with iodides, and given for periods of several weeks at a time, affords the most reliable of all methods of medicinal treatment. Cod-liver oil always aids this combination, and alone is of much value in most cases. Drugs for the relief of pain, as salicylates, aspirin, salol, &c., are very uncertain in their action.

Local treatment by counter-irritation, capsicum, cantharides, and the cautery, or soothing liniments to relieve pain, are also of little value in most cases: any value derivable from them being probably caused by the friction or movements employed in their use.

Hot sand, the local application of steam or of dry heat (250°–300° F.) by any of the electric or gas machines, give often marked but very temporary relief. Electricity has been tried in every form, but beyond sometimes relieving pain it has no effect upon the progress of the case; combined, however, with the next agent, electricity is always of value. Massage with passive movement is unquestionably the most reliable of all agents in the treatment of this almost hopeless joint-disease; it is best used in conjunction with the various forms of baths (general or local), and especially with the Scotch douche (alternating cold and hot douches), or with the hot

douche alone as at Aix-les-Bains and Harrogate, or with the peat bath as at Strathpeffer. Massage and passive or forced movements in many cases cause considerable pain, but even at the risk of aggravating or exciting neuritis they should be persevered in for many months, as they afford in some cases the only hope of breaking down adhesions polishing the roughened joint-surfaces and preventing that locking of the articular surfaces by osteophytes which ends in the utter uselessness of the affected limb. They can be employed during the winter months at home whilst the patient is in a vapour or Turkish bath.

Ewart reports satisfactory results by what is practically a new method of treatment, which consists in the application of an elastic tourniquet to the limb after stroking and elevation, so as to produce a local anæmia for about one minute: the pressure is then suddenly removed, so as to produce arterial flushing and suctional drainage (*Lancet*, August 13, 1904).

ASCARIS LUMBRICOIDES

This nematode, known as the round-worm, is common in the upper part of the small intestine in children, and sometimes in adults. It resembles an earth-worm, the female measuring from 8 to 16 inches and the male from 6 to 10 inches. It is partially transparent, of a glistening pink colour, tapering at both ends, and marked by closely set transverse striæ. The mouth has three lips bearing papillæ, and in the body of the female the tortuous uterine tubes, resembling white threads, may be seen distended by millions of ova which appear in the fæces as ovoid bile-stained bodies about $\frac{1}{300}$ inch (·075 mm.) in their greatest diameter, with the shell covered over by a mammillated albuminous sheath. The contents appear granular. Outside the body these ova do not show signs of embryos for months; the ova are probably swallowed in drinking-water, and after the digestion of the outer shell the young parasite is set free in the stomach or intestine. An intermediate host, as in the case of the tapeworm, is not necessary. From one to a hundred may be lodged in the alimentary canal at one time. They sometimes creep back into the stomach, and the writer has seen one pulled out through the nostril after vomiting, and he has seen one wriggle out through a biliary fistula at the umbilicus of a child. They are liable to invade the bile and pancreatic ducts, and have found their way into the glottis, causing serious trouble.

Symptoms.—The phenomena produced by their presence are very vague or ambiguous. All sorts of nerve-ailments are attributed to them; but it is tolerably certain that large

numbers may infest the bowel without causing any ill-effects of any kind. The writer, to test the value of the statements made upon this point, administered santonin in a routine way to large numbers of children brought to the extern of a children's hospital suffering from minor surgical, cutaneous, and other trifling ailments; the results led him to believe that the parasite is probably always present in every child at some period of its life, and that it never causes any symptom except in the rare cases when it may block up the bile-ducts or cause local bowel irritation or obstruction. The popular idea that its presence causes itching at the anus and at the nose, grinding together of the teeth, and general convulsions, appears to have little or no foundation in fact.

Treatment.— Santonin kills the worm when properly administered. One dose is generally sufficient, and if the drug be given in powder mixed with a spoonful of castor oil serious symptoms never appear. The dose for a child three to six years is 2 grs., adults may get 5 grs. It may be given at bedtime, and, if necessary, a further dose of oil or calomel may be given in the morning. A minute portion of fæces will almost always show ova, when covered with an object-glass and examined by a low power, if any worms exist in the intestine.

The same treatment is applicable to Ascaris mystax, a very rare and much smaller round-worm.

The prophylactic treatment for the round-worm will consist in the boiling and filtering of all drinking-water and the thorough cleansing of all vegetables eaten uncooked. The writer believes that the parasite is propagated by children eating fruit which has fallen and lain upon the earth under the fruit-bushes.

ASCITES

This is not the name of a disease: it is merely the term used to denote an effusion of fluid—serous, fibrino-serous, or chylous—into the peritoneal cavity. The causes are:

1. Obstruction to the portal circulation *within* the liver, as seen in cirrhosis of that organ and in long-standing passive congestion caused by mitral disease and lung-affections, in rare forms of primary cancer of the liver, and in syphilis affecting the liver itself.

2. Portal obstruction taking place *outside* the liver by the pressure of cancerous growths and tumours on the vein at the transverse fissure, and also by thrombosis of the portal vein.

3. Disease of the peritoneum, as chronic inflammation, especially the forms of inflammation associated with cancer, sarcoma, and tubercle, when they invade the abdomen, and

the peculiar peritoneal condition which is induced by innocent ovarian tumour and enlarged spleen.

4. The ascites may be part of a general dropsy caused by renal disease or cardiac failure.

5. Chylous ascites caused by rupture of the thoracic duct or lacteals.

Symptoms and Signs.—The abdomen uniformly enlarges as the fluid accumulates, and in fat subjects small amounts can only be demonstrated with difficulty by very careful percussion in different positions of the body. When the sac is moderately full the signs are distinctive on inspection, palpation, and percussion.

In the erect position the abdomen protrudes forwards and downwards ; when the patient lies upon his back the front is flattened, and bulging is evident at the sides. With the sac well filled the barrel-shape is less altered by position and the entire abdomen is tense, the costal arch and ensiform being turned outwards, and the umbilicus may protrude. The skin is tense and glistening, and white lines—the lineæ albicantes—appear above Poupart's ligament ; the superficial veins are flattened out and very obviously visible. Breathing becomes shallow and difficult, and the heart is displaced upwards.

By placing the fingers in contact with the skin over one flank and smartly tapping the opposite side with the finger-tips of the right hand, a distinct wave of fluctuation can be felt by the left hand, and it can often be seen by the eye. A pendulous fat abdominal wall gives a false sense of fluctuation to the hand upon palpating in this manner ; the fallacy is excluded by asking the patient or an assistant to make pressure with the edge of a thin book, or with the ulnar margin of the hand laid along the middle line of the abdomen, when the fluctuation-shock does not occur unless fluid be present.

A very valuable method of palpating, known as ' dipping,' is to place the finger-tips at a right-angle to the skin, and by a suddenly executed jerk press them downwards as the patient lies upon his back, so as to displace the fluid immediately beneath them. In this way a solid organ like the liver or a tumour may be unmistakably felt.

Percussion gives generally reliable results, because, the fluid being free in the peritoneal cavity, it will change its position as the position of the patient's body is altered. Thus, in the erect position dulness on percussion in the middle line may extend to near the ensiform ; upon placing the patient on his back the fluid occupies the flanks, which are dull, whilst the intestines float and give a clear resonant note in the umbilical region. Upon turning the patient on his side the uppermost flank becomes tympanitic, and this in its turn

becomes dull when placed undermost. By placing the patient in the knee-elbow position, very moderate amounts of fluid may be demonstrated. There are three fallacies to be guarded against in examining an ascitic patient: the mesentery may be so shortened by peritonitis that the intestinal coils cannot float up in contact with the abdominal wall ; the colon may be so distended by gas as to give clearness in one or both flanks ; the fluid may be encysted by adherent omentum or intestine.

The peritoneal sac may contain as much as forty-five pints of liquid.

Ascitic fluid is usually clear and transparent and of a pale yellowish-green colour. When a simple transudate, its s.g. varies from 1008 to 1015 ; when it is an inflammatory exudate it is 1015–1024. Albumin is present in all cases, and sometimes blood ; occasionally urea and cholesterin are found ; the fluid from inflammatory action is fibrino-serous, has a more marked tendency to coagulate, and is richer in albumin. The reaction is nearly always alkaline, but a neutral or faintly acid reaction has been very occasionally observed.

In the chylous variety, fat is always present in large amount ; but a variety is recognised as 'fatty' which owes its slight milky appearance to the fatty degeneration of endothelial cells, and under the microscope it shows large fat-globules. The true chylous ascitic fluid has its fat in the form of an emulsion—the molecular basis of chyle.

A typical case of chylous ascites is fully recorded and figured by the writer in the 'British Medical Journal' (May 1885). The case was unique, inasmuch as the autopsy revealed a minute perforation in the walls of the receptaculum chyli, which followed dilatation of the duct due to the plugging by a tubercular nodule higher up (in its middle third). The perforation had obviously occurred a considerable time before death and permitted all the chyle to escape into the peritoneal cavity during life. Fifteen gallons of apparently pure chyle were removed from time to time by tapping, sometimes as many as twenty-two pints being withdrawn at once. The fluid, upon being submitted to careful analysis by Professor Matthew Hay, was found to consist almost entirely of pure chyle, which, owing to the lowness of the percentage of albumin present, was obviously free from any marked dilution with serum produced in the peritoneum. The s.g. of the fluid varied from 1015 to 1020, and resembled a sample of rich milk from the cow, showing a layer of thick cream, upon standing, which measured one-eighth of the height of the column.

Diagnosis.—1. This will consist in a differentiation of ascites from the diseased conditions resembling it. The greatest difficulty will be in the case of a large thin-walled

ovarian cyst. The history will probably show that the enlargement began in one side, gradually encroaching on the middle line before filling the abdomen. Even when large, a certain amount of asymmetry may be demonstrated by measurements on each side between the iliac spine and umbilicus. The fluid, not being free, presents no marked percussion-differences on altering the patient's position, and the intestines are crowded into the flanks, giving a tympanitic note, whilst dulness in front is the rule. The abdomen measures more below the umbilicus than above it, whilst in ascites the greatest girth is at the umbilicus, or just above it ; in ovarian cyst the line between the pubes and the umbilicus is longer than the line between the sternum and this point, the reverse being generally the case in ascites. When peritoneal effusion accompanies a thin-walled cyst, the difficulty is greatly increased ; a vaginal and rectal examination may reveal the connection of the tumour with the uterus, which it displaces to the opposite side.

Large *renal*, *hydatid*, and *pancreatic* cysts are as a rule easily eliminated by palpation and percussion, and in cases of doubt an examination of their contents settles the point. The fluid from an ovarian cyst is generally much more viscid, dark-coloured, and grumous, with high specific gravity.

A *distended bladder* may simulate ascites ; but the passage of a catheter will instantly settle the matter, and it is always well to make this a routine procedure before tapping an ascites. *Pregnancy*, with excessive liquor amnii, is eliminated by the history and by a careful search for the other signs of utero-gestation.

2. The diagnosis of the cause of the ascites should always be made. The history of chronic alcoholism generally settles the question of *hepatic cirrhosis*, and the clinical picture of the muddy icteroid skin and tinged conjunctiva, with venous stigmata on the cheeks, is almost distinctive. Often in the earlier stages the enlarged and firm organ may be felt below the arch by ' dipping ' the fingers so as suddenly to displace the layer of fluid ; the enlarged spleen can generally be made out, and there is constant nausea and often hæmatemesis and bleeding piles. Ascites the result of *renal dropsy* is never abundant, and the signs of renal disease are so obvious that a difficulty in diagnosis never occurs.

Mitral lesions give a distinctive history, and the cardiac signs are obvious. *Cancer* of the liver, gall-bladder, and pancreas may generally be inferred by the progress and history of the case ; the ascites in these cases being caused by enlarged glands or growths at the transverse fissure of the liver. Jaundice is very often present, and is always progressive in character and deepens with the wasting. It is, moreover, very often associated with pain. Where the ascites is the result of a peritonitis caused by a general carcinomatous invasion of the

serous membrane, the wasting is very rapid. The serous reduplications and ligaments attaching the abdominal organs are shortened in these cases, and the writer has noted a very characteristic sign about the umbilicus: this may be drawn inwards and surrounded by a narrow zone of tissue, as if a small steel ring were inserted under the skin. It is probably caused not by a deposit of new-growth, but by the tautness produced by the shortening of the liver-ligament, and it does not occur in simple or tubercular peritonitis.

The ascites caused by *tubercular peritonitis* is as a rule easily diagnosed. It is most frequently met with in young subjects. There is considerable pain and interference with the bowel, the most characteristic disturbance being diarrhœa of a painful kind, alternating with periods of constipation. Masses of enlarged glands or of mesentery, matted with coils of bowel, may be often felt extending like a roll of wide bandage across the middle of the abdominal cavity. Evidence of phthisis or of tubercle of lymphatic glands in the neck, accompanied by wasting, hectic fever, and other characteristics of tubercular mischief, are often present.

Syphilitic affections of the liver producing ascites are rare, but the history and other manifestations of the disease should throw light upon doubtful cases; it should be remembered that the marked thickening and deformity of the organ by gummata and perihepatitis, so liable to be mistaken for cancer, are not associated with the rapid and progressive emaciation always found in malignant disease.

Ascites of any magnitude, the result of a simple or idiopathic inflammation of the peritoneal surface, probably never exists; its presence may be suspected by a process of exclusion.

The history of the onset in all cases of *perforative peritonitis* followed by ascites is so definite that a difficulty in diagnosis does not arise.

Prognosis.—This of course will depend upon the nature of the cause of the ascites, and need not be discussed, as most of the conditions are incurable; but even in the tubercular form recovery not infrequently follows operative treatment. Cirrhosis almost always ends fatally, but in some cases early tapping has been followed by permanent improvement. Syphilitic perihepatitis and gummata, causing ascites, may yield to mercurials in combination with large doses of iodides.

Treatment.—The treatment of the original cause need not be here discussed; it will be found mentioned under its own heading.

In dealing with the accumulation of ascitic fluid, if moderate, before resorting to tapping, the action of saline cathartics, diuretics, diaphoretics, absorbents, and blistering may get a fair trial. When pressure-symptoms become marked, the

fluid must be evacuated by the trocar and cannula. The patient should be placed in an easy sitting posture, the bladder having been previously emptied ; a long binder, placed around the abdomen so that the ends on each side are free, is used with the view of exerting pressure to keep up the tension as the fluid flows. The skin in the middle line, half-way between the pubes and umbilicus is sterilised, and at a spot where absolute dulness is obtained on percussion a small incision is made and the subcutaneous tissue reached ; through this a trocar and cannula are quickly and firmly pushed home. On the withdrawal of the trocar the fluid freely flows, and the tension within the abdomen is kept up by pulling on the ends of the binder. It is well to have an opening in the binder corresponding to the site of the incision. Upon withdrawal of the cannula a pad of antiseptic lint is placed over the opening and a fresh binder wound tightly around the shrunken abdomen. Subsequent constant pressure by suitable bandaging sometimes retards the progress of the effusion. Southey's tube may be employed, and it may be left in for many hours, till the sac is completely drained.

In cirrhosis and other forms of portal obstruction the operation of suturing some omentum to the abdominal wall with the view to establish anastomosis may be undertaken, and the recent results are most promising in cases of cirrhosis. (See under ' Liver, Cirrhosis of.')

ASTHMA

This is a neurosis characterised by recurring attacks of dyspnœa caused by spasm of the muscular walls of the bronchioles, associated with an erectile or turgescent condition of the bronchial mucous membrane.

Etiology and Pathology.—The disease may occur at any age ; it is twice as common in men as in women ; and often an hereditary history of epilepsy, megrim, or of the hereditary transmission of the tendency to asthmatic seizures may be obtained. In children it may follow pertussis, and in adults may appear as a sequela of repeated bronchial attacks or of hay-asthma.

When the diseased condition has become established the exciting causes are numerous ; thus, certain odours, irritating dust, nasal polypi, severe mental emotion, dyspepsia, and uterine disturbance precipitate an attack. But of all the exciting causes none are so potent and constant as bronchial catarrh ; this, no matter how induced, generally brings on an attack in the asthmatic patient. In a large percentage of asthmatics, catarrh of the bronchial tubes is always more or less present, and hence by some it is regarded as a necessary

part of the disease. Many cases of the disease will, however, be found perfectly free from all traces of bronchial catarrh between the attacks, and in these any catarrhal seizure is soon followed by an asthmatic paroxysm. Even in asthmatics who continually suffer from symptoms of chronic bronchitis any exposure followed by a fresh catarrh determines the advent of a severe spasmodic seizure.

Of late years much attention has been directed to the condition of the nasal passages in asthmatic subjects, and unquestionable relief has so often been obtained by the removal of adenoids, enlarged tonsils, polypi, and hypertrophic thickenings of the mucous membrane and other abnormal obstruction to the air-way in the nose, naso-pharynx, and pharynx, as to give considerable weight to the view that the paroxysms may be traced to a reflex action having its source in these portions of the respiratory tract. Wilkinson regards the reflex spasm as due generally to some irritant of an aërial source, as the emanations from domestic animals or birds; and the irritation caused by breathing the dust from feather-beds has been long recognised as the source of attacks in individuals exhibiting this idiosyncrasy. Whilst the asthmatic paroxysm is generally accepted as explicable upon the theory of spasm of the muscular element in the bronchioles, Hare insists upon a vaso-motor mechanism of the attack being the most important, and he points out that vaso-constriction occurring in other parts of the body leads to vaso-dilatation of the bronchial area; hence the asthmatic seizure is often due to, or aggravated by, cold applied to the skin. The theory of the asthmatic paroxysm being directly due to turgescence of the bronchial surface has many advocates, and probably both spasm of the tubes and turgescence of the bronchial mucosa may be factors operating together at the same time. The phenomena of cardiac asthma are partly explained by the turgescence theory, but only in some cases is there evidence of involvement of the bronchial surface. In this connection Hadley draws attention to the *immediate* relief sometimes following chloroform-inhalation and the injection of morphia as evidence in favour of the theory of spasm, turgescence not being conceivably relieved in such a rapid manner. The theory of the origin of the spasm being due to mechanical obstructions in the bronchi of a true inflammatory nature has less to be said in its favour, since, though the attack is often excited by bronchial catarrh, the secretion is usually very scanty; but the presence of the spiral sputa lends weight to this theory. Spasm of the diaphragm and of the intercostal muscles has been credited with the production of asthmatic seizures. The reflex source of the spasm has been demonstrated in some instances as originating in the stomach in cases of over-eating and dyspepsia, and in the uterus, ovaries,

and rectum in other cases; but abnormal conditions in these viscera can only be regarded as exciting causes which bring the established neurosis into evidence. In rare cases the centric origin of the spasm is seen in brain-lesions involving the vagus-roots.

Symptoms.—In a typical case the patient retires to rest in his usual health, and wakes up after a few hours' sleep to find himself burdened by considerable dyspnœa. He acutely feels the want of air, and sits up in bed or stands on the floor, seizing some firm support with his hands so as to get a fixed basis for his extraordinary muscles of respiration, as the efforts to breathe become more laboured or violent. His countenance, at first pallid and anxious, soon shows signs of cyanosis, and cold perspiration appears on his forehead. These, with the gaping mouth, dilated nostrils, retracted head, and painfully laborious breathing, form a picture which, when seen for the first time, always creates alarm.

On inspection of the thorax, all the respiratory muscles are seen in violent action; the chest-walls seem fixed in a position of full inspiration, and the diaphragm is almost immovable and lowered; the difficulty is obviously in expiration, which is loud, wheezy, and prolonged, followed immediately by a short abortive inspiration which permits of the entrance of very little air. The pulse is quick and small, the number of respirations only slightly increased, and the temperature is normal.

The expiratory dyspnœa is so great that the patient is unable to speak or cough. The attack may last from half an hour to half a day or more in length, and improvement generally sets in after coughing becomes established. This is at first very hard and dry, but gradually some peculiar expectoration appears and the attack slowly subsides.

During the paroxysm, percussion gives a clear resonant note, and auscultation reveals absence of the true respiratory murmur and the presence of sibilant and sonorous rhonchi of all varieties of pitch and timbre. One of the most characteristic signs of asthma is the ever varying character and shifting distribution of these wheezing sounds during the paroxysm; they change their quality and position whilst the auscultator has his ear to the chest. After the attack passes off, the moist bubbling sounds of catarrh are heard, until expectoration ceases. The expectoration is very characteristic: small, round, whitish masses like tapioca are coughed up. These, if spread upon a piece of glass placed over a black surface, are found to consist of convoluted threads in the form of spirals, visible to the naked eye. Under the microscope these spirals are seen to consist of a central thread of mucin wound round by delicate fibrils, amongst which are entangled eosinophile leucocytes and granules. The spirals

are supposed to be casts of the finer bronchioles, and Osler thinks that their corkscrew character may be given to them by some rotatory motion, the result of ciliary action. They are known as Curschmann's spirals, and are believed to be distinctive ; but they have already been found in other diseased conditions of the lung. The peculiar bodies known as Charcot-Leyden crystals are also found in association with the spirals ; they are phosphates of some organic base like spermin, and resemble those found in the blood in leukæmia.

An examination of the blood reveals great increase of the leucocytes of the eosinophile type, which is a useful diagnostic point in the elimination of some forms of pulmonary œdema of obscure origin.

The attack may be followed by others at regular intervals of several hours or days, during which the patient is quite well ; more frequently, however, the wheezing left by one attack has scarcely passed off before another attack the following night supervenes. Some of the severe paroxysms may be followed by others so slight as not to inconvenience the patient, and many asthmatics get premonitions of a seizure long before it comes. In some cases the attacks are divided by intervals of years.

Repeated attacks generally produce emphysema, and are, as already stated, often associated with chronic bronchitis, so that the chronic asthmatic presents in his person characteristics almost as distinctive as those met in any disease. He is thin, stooped in his high shoulders, has a dusky face with a heavy listless eye surrounded by pouches of œdematous tissue, and his head tends to fall forwards, so that his chin rests on his sternum, the entire aspect indicating weariness even when in the sitting posture. His gait is also noticeable, and any exertion tells upon his embarrassed breathing apparatus, wheezing, with cough and feebleness of voice, being then brought into evidence.

Diagnosis.—The attacks of shortness of breath occurring in heart-disease and renal affections, and known as *cardiac* and *uræmic* asthma, do not present the features of the *laboured expiratory dyspnœa* so marked in spasmodic or bronchial asthma. The breathing in these cases is of a gasping or panting kind, and the respiration-rate is generally much increased. When asthma occurs in patients the subject of renal or cardiac disease, and when severe dyspnœa accompanies an acute bronchitis, the diagnosis is difficult ; but the history of the case and the presence or absence of the spirals in the sputum will generally solve the problem. The *dyspnœa* of laryngeal obstruction, with its characteristic voice-changes, is easily differentiated, and so also is that produced by the pressure of aneurismal or other growths ; in the latter the physical signs reveal the nature of the difficulty in breathing

which is always more or less continuous, though liable to severe exacerbations on movement.

Prognosis.—There is no danger to life in the paroxysm, and upon the whole life is little, if any, shortened in chronic asthmatics, unless in those where chronic emphysema and dilatation of the heart supervene in a marked degree. Complete recovery is common in young subjects, but when attacks begin after middle life they seldom leave the patient except for longer or shorter intervals. The prognosis is very much influenced by the facilities which the patient can command for changing his climate, occupation, or environment. The writer has seen several cases where the attacks alternate with attacks of migraine, and in these the prognosis is better, he thinks, than in those where no obvious cause of attack is apparent.

Treatment.—In pages 64–70 of the author's 'Dictionary of Treatment,' the details for the management of this disease are set forth at length. The relief of suffering in the paroxysm is best accomplished by inhalation of chloroform, amyl nitrite, ethyl iodide, the fumes from burning nitre-paper or from any of the various powders containing stramonium and nitre. One moderate dose of morphia hypodermically seldom fails to give relief. Where the dyspnœa is not so great as to prevent the patient smoking the chopped-up leaves of datura tatula or stramonium, these may be substituted for the burning powders. All the agents which relieve the attack may be used to prevent it, if resorted to as soon as the patient gets warning of its advent; chloral hydrate with atropine is very efficacious if given early.

Of drugs which have a distinct curative action, or which seem to diminish the tendency to attacks, the best are iodides and arsenic, and these can be combined generally with advantage or alternated in courses of about one month each in full doses. Where bronchitis is present expectorants like ipecac. and lobelia may be given alone, or in combination with the iodides. Where iodides fail, marked benefit may be still obtained by the administration of free iodine in tincture form. Cardiac tonics should be combined with the iodine treatment where there are evidences of dilatation, and *moderate* doses of strychnine are valuable under such conditions: large or full doses may precipitate an attack.

Diet is of great importance, and the rule should be insisted upon that the patient takes his meals with regularity every three or four hours during the day, so that he shall be enabled to retire to rest at least three or four hours after his last meal. The late heavy dinner must be abolished.

The most important consideration in the treatment of asthma is the study of the causes in the patient's environment which lead to the attacks; these are often inscrutable, but by

observation and experiment in an empiric way valuable guides can always be obtained. One patient, for example, may be able to live free from attacks if he change his residence but a few miles from the scene of his constant illness ; some can enjoy immunity in the smoky city when a change to the seaside is immediately followed by seizures ; and there are few cases in which the patient himself has not made the discovery of some region in which he enjoys comparative freedom from attack, and he should be urged to reside permanently in such a locality when possible.

Climate is of vital importance, especially in the case of the young, where there is bronchial catarrh, and such should always, when possible, be advised to winter in a warm equable climate. It is needless to remark that the patient should never expose himself to the risk of coming into contact with the emanations which he has learned to recognise as the cause of his seizure. The nose and naso-pharynx must be examined, and enlarged tonsils, adenoids, polypi, or other source of irritation removed. The writer has seen marked improvement follow the extraction of numerous foul decaying teeth, and he believes that marked errors of refraction may accentuate the tendency to attack. Relief often follows cauterising the septum as advocated by Dr. Francis of Brisbane, but in the majority of cases the attacks return. The writer has seen such benefits follow its use in the hands of Dr. Joseph Nelson that he often resorts to it as a preliminary routine in severe cases.

ATAXIA, LOCOMOTOR, or TABES DORSALIS

This is a diseased condition caused by a degeneration of the posterior nerve-roots and of the posterior columns of the cord, manifesting itself in a characteristic group of symptoms amongst which incoördination, defective sensation, trophic disturbances, peculiar pains, and ocular phenomena are prominent.

Etiology and Pathology.—The diseased process, according to Gowers, is confined to the sensory neurons dependent upon the posterior ganglia, the motor neurons never suffering in pure tabes. The involvement of the spinal ganglia first shows itself in degeneration of the posterior nerve-root. The fibres passing into the cord from the posterior root conveying sensations from skin, muscles, &c., divide into three sets :

1. Fibres forming the spinal arc. These terminate in arborisations round the motor cells in the anterior horn ; hence their degeneration causes a gap in the arc, which explains the loss of the knee-jerk phenomenon, and the loss of tonus, the sensibility of the muscles having become diminished or absent.

2. Fibres which pass upwards through the column of Clarke on their way to the cerebellum; their degeneration cuts off the cerebellum from peripheral impulses, and this affords an explanation of the interference with equilibrium.

3. Fibres which, on their way to the optic thalamus, pass upwards forming Goll's column; degeneration of these cuts off the direct sensory path to the opposite cortex. This, in conjunction with the destruction of the other two sets of fibres and the afferent nerves shown by Sherrington to accompany the fibres of the motor nerves, produces the typical condition of ataxia characteristic of the disease, all the centripetal impressions from muscles, joints, and tendons having been cut off.

Little, if any, change is to be found in the spinal ganglia themselves, because the first portion of the afferent neurons to suffer would be the arborisations farthest from the influence of the ganglion-cells. Thus it would seem almost certain that the sclerosis of the posterior columns is a secondary degeneration. When the degenerative change proceeds in the peripheral direction from the ganglia the sensory nerves become involved.

The degeneration generally begins in the posterior nerve-roots in the lumbar region; it may, however, start in the cervical region, constituting cervical tabes.

The cord appears flattened, and the pia arachnoid is often thickened and adherent, especially upon the posterior surface. The posterior roots and the posterior columns are atrophied and increased in consistence, and are of a translucent grey colour. In early cases the atrophic changes are best marked in or confined to the middle portion of the posterior columns; but in long-standing cases the entire bulk of the posterior external columns is invaded in the lumbar region; as a rule, however, the endogenous fibres escape. In the cervical region, the column of Goll, the posterior median, or the postero-internal exhibits the degeneration. The histological examination shows the degenerative changes to consist of the destruction of the myelin sheaths, with wasting of the axis-cylinders and great increase in the neurogliar tissue, which explains the obvious sclerosis. The degenerative changes are seen also in the posterior nerve-roots as well as in the systems which, as already explained, constitute the prolongation upwards of the fibres entering through these roots. The pathological changes are usually therefore accepted as of the nature of a true 'system' disease.

In the cerebrum, the optic nerve and the nerves supplying the eye-muscles and the tongue, together with their nuclei, may be involved, and occasionally the pneumogastric, glosso-pharyngeal, fifth, and ear-nerve may suffer.

The disease is about ten times more common in males, and generally shows itself about forty. A history of syphilis

is obtainable in about nine cases out of every ten, though other causes are believed to be at work, as sexual excess, trauma, overwork, exposure to extreme cold and damp, &c. A form known as 'Juvenile tabes,' due to heredo-syphilis, is also recognised, of which the writer has recently seen a case under the care of Dr. McQuitty. Though so intimately associated with syphilis, it must be remembered that the disease certainly is not simply a manifestation of the tertiary stage of this affection, since it is uninfluenced by the drugs which exert such marked specific action in syphilis. It belongs to the *parasyphilitic* or *quaternary* stage of the disease. In all probability the starting-point in the altered nutrition of the spinal ganglia or roots is induced by some chemical poison secreted by the syphilis virus in the long-past secondary stage of its existence. It may be the product of a microbe which invades the system in company with the syphilitic micro-organism, or, as the writer conjectures, the disease may be owing to the absence of such a microbe. If we assume that in a case of ordinary syphilitic infection a microbe accompanies the specific one and modifies its ultimate action, the absence of such organism might determine the advent of ataxia at a later stage. It is to be remarked in this connection that the most typical examples of ataxia have followed in cases where the local chancre was of the mildest and most insignificant character. This conjectured dual nature of the syphilitic infection might be used as an hypothetical explanation of the relative frequency with which ataxia is met in the case of husband and wife (conjugal tabes). The period of advent of ataxia varies from about seven to fourteen years after the syphilitic invasion, but it may be a little earlier or later.

Orr and Rows hold the view that tabes is the result of the absorption of toxins from peripheral septic foci, the toxins passing up the perineural sheaths without injury to the nerve-fibres till the posterior roots are reached. They conjecture that the bladder may be the source of such peripheral septic foci.

Ford Robertson maintains that tabes is the result of the invasion of a diphtheroid bacillus—the *Bacillus paralyticans*—in persons whose vital resistance is lowered by syphilis, alcohol, or over-feeding with nitrogenous foods. The micro-organism which he has isolated is identical with that found in general paralysis of the insane, and he believes that it enters the body through the pulmonary and alimentary tracts, but that, owing to the activity of the polymorpho-nuclear leucocytes, it is impossible for it to live upon a healthy mucosa.

The influence of fatigue and over-exertion cannot be eliminated in the etiology of tabes; and recent researches (see under

'Paralysis, General, of the Insane') go to prove that when in the parasyphilitic stage the higher cerebral centres are over-exercised general paralysis follows; but when the spinal paths are subjected to the strain tabes is the result. Hence the significance of the terms 'spinal' and 'cerebral tabes'—the parts submitted to the greatest strain succumbing to the degenerative process.

Symptoms.—In typical cases three stages of the disease may be met with.

1. *The pre-ataxic stage.*—In this the first symptom noticed by the patient may be the strange pains which are almost characteristic of the affection—the so-called lightning-pains. These like a series of electrical shocks pass through the lower extremities or other portions of the body at irregular intervals. They come on suddenly in paroxysms, following each other for a few minutes, and the attack may last for hours, after which the patient may enjoy freedom for weeks or months. In rare cases, as recently pointed out by Gowers, the lightning-pains may constitute the only symptom of the disease. If examined at this stage, one of the most reliable signs of the disease is almost invariably found. This, known as Westphal's sign, is the absence of the knee-jerk; it always precedes the ataxia. The ankle-jerk, according to Babinski, may disappear before the knee-jerk.

Ocular signs of scarcely less importance are also to be found. The Argyll-Robertson pupil is equally important, though not invariably present. It is a reflex iridoplegia; the pupil does not contract when exposed to light, though the power of contraction is demonstrated when the patient looks at a near object—*i.e.* when he accommodates.

The pupils are generally contracted, sometimes to pin-points. Various other eye-phenomena are observable; thus, *temporary* paralysis of all or any of the eye-muscles, with double vision, is not uncommon. Of more importance, however, is atrophy of the disc, which may even be the first sign of the disease in some cases; it occurs in about every seventh case of the affection; and after dimness of vision, colour-blindness, and diminution of the field of vision, the patient may find himself totally blind. When the disc atrophies early, the symptoms of incoördination may be indefinitely postponed, and the disease may remain in this stage for years; deafness is not uncommon. In the pre-ataxic stage various neuralgic, sensory, and other symptoms may be present which are to become more marked in

2. *The second, or ataxic stage.*—Here progressive motor phenomena with sensory disturbance dominate the clinical picture. The first symptom is generally the unsteadiness of the patient, especially when in the dark he stands or attempts to walk. There is no paralysis and the muscle-reactions are

normal ; but the subject of ataxia is unable to coördinate his muscles harmoniously, so that ordinary movements when attempted fail in their intention, or become markedly clumsy. Romberg's sign becomes soon well marked. This is elicited by placing the patient in the erect posture with his feet close together ; when he shuts his eyes, the body-equilibrium is lost, and he sways to and fro, and falls unless supported.

At this stage his gait is characteristic : he starts unsteadily with his legs somewhat apart, lifting his feet too high and jerking out the limb, which comes down with a sudden stamp caused by the entire sole striking the ground all at once. If he attempts to stop suddenly or turn, he staggers and may fall, and it is observed that he leans his body forwards and watches carefully his legs and the ground upon which he treads. He cannot stand on one leg : he requires the use of a staff, and soon a staff in each hand becomes necessary to steady his body when in motion.

At a later stage the incoördination ascends to the arms ; and though there may be no appreciable muscular weakness, the patient may be unable to execute the movements necessary in the operation of dressing himself, especially such movements as he cannot guide by his sense of sight. Gradually some reduction in muscular power supervenes, and permanent ocular or tongue-paralysis may now show itself, and hemiplegia may occur in rare cases. Laryngeal palsy is a serious complication ; it consists of bilateral abductor paralysis. Anæsthesia and sensory symptoms are marked, the lightning-pains continue at irregular intervals, and all sorts and degrees of sensory disturbance are experienced, as 'pins and needles,' formication, numbness to complete anæsthesia of the soles, which causes the patient to feel that he is walking on wool. A true *tabetic neuralgia* has been described by Gowers which may exist without ataxia. Sensory impressions are markedly delayed or distorted ; a pin-prick in the leg may not be felt at all, or only appreciated after a considerable interval, or referred to another region (*allocheiria*). The muscular sense becomes lost, so that the ataxic patient is not conscious of the position of his limbs.

Bladder-symptoms become prominent. In the first stage the calls to urinate may be frequent, and the stream of urine may be long noticed to be sluggish ; now expulsion becomes difficult, dribbling at the end of micturition is noticed ; finally incontinence or retention supervenes, and in the later stages many, if not most, ataxics die from bladder-trouble.

Visceral symptoms are the rule, and they constitute the group known as the 'crises.' Pain and disturbances of function of a severe paroxysmal kind attack the stomach, larynx, heart, intestine, anus, urethra, kidney, or nares. Thus, in the gastric crises, pain beginning in the epigastrium and

radiating backwards to the spine and upwards towards the chest comes on in spasms of great intensity, accompanied by paroxysmal vomiting, which may last for many hours before suddenly departing, to return after regular short or long intervals. Sometimes the sudden explosive paroxysms of vomiting are not associated with any pain, and in some attacks the pain is unaccompanied with vomiting or nausea. In the laryngeal crisis there is true laryngeal spasm with stridor and dyspnœa.

These crises may be present from the beginning of the disease in the ataxic period, and may be the first symptoms to attract attention; they are supposed to correspond in some way to the attacks of lightning-pains.

The trophic changes are of great interest: minor signs are always present somewhere, as local sweating, shedding of the nails and teeth, herpes, &c. The perforating ulcer of the foot is a most characteristic phenomenon, commencing as a suppurating corn under the prominence of the first or fifth toe; the ulcer gradually eats its way through to the dorsum of the foot. It appears to have no 'action' about it, and seems like a sore on a cadaver; a probe may be made to pass through the foot from sole to dorsum, and the patient seems not to have any sensation in the neighbouring zone of tissue; nevertheless, under local electric or other treatment it may completely heal.

The bones and tendons get brittle and may snap with little provocation; but it is in the joint-affection (Charcot's joint-disease) that the most surprising results are to be sometimes seen. A huge painless swelling suddenly appears about a joint like the hip, or knee, which a few minutes before seemed to be normal, and in a few days, as the effusion has disappeared, the joint may be found so completely disorganised as to resemble a flail. The writer has seen with Dr. F. E. MacFarland, a patient who had gone to bed after rubbing his limbs with a towel, as was his usual custom; some minutes later he accidentally discovered a large swelling about his buttock. When seen an hour afterwards the effusion around the hip-joint must have represented many pints; it was as large as a human head and absolutely painless. The patient was certain that there was nothing wrong when he gently rubbed the part a few minutes before, and he would not have been aware of its existence unless his hand had come in contact with it in adjusting the bedclothes. The joint was greatly disorganised before the end of a week, when the œdema had disappeared. Generally the joint-changes are not so rapid as in the above case, and the typical Charcot's joint of ataxia may closely resemble that found in rheumatoid arthritis, owing to osteophytic outgrowths in the capsule with extensive destruction of cartilages and of the articular surfaces of the bone. This is not rarely seen in the foot, where the joints at

the inner part of the sole form a large projecting bony mass filling up the arch and projecting on the dorsum of the foot. The characteristic joint-changes may occur early in the disease ; suppuration does not appear as part of the disorganising arthropathy. The osseous tissue becomes rarefied and permits a freer passage to the X-rays.

3. *The third, or paralytic stage.*—In this all the symptoms deepen, and there may be a genuine paraplegia, according to Ormerod. The ataxia is so marked that the patient is unable to stand and he is compelled to spend his time in an easy-chair ; later on he becomes permanently bed-ridden. Incontinence or retention of urine sets up bladder and kidney mischief, which carries him off if he become not the victim of bedsores, pneumonia, or cerebral apoplexy. The sexual function may be over-active in the early stage ; it is generally diminished in the second and entirely absent before the paralytic condition supervenes ; but there are exceptions in all stages.

The course of the symptoms and duration of the disease are most variable. One patient may become helplessly ataxic in a year, whilst another may continue able to walk about and attend to business for a quarter of a century. The pains and visceral crises may disappear in some cases after the establishment of incoördination, and in many instances the march or progress of the symptoms may become spontaneously arrested for many years.

Diagnosis.—Even in the early stage this is not difficult. The combination of the important signs are distinctive when present—*i.e. Absence of knee-jerk, Argyll-Robertson pupil, lightning-pains, visceral crises, disc-atrophy*, and *temporary eye-palsies*, followed later by *incoördination* as tested by Romberg's sign. In the majority of cases the presence of Westphal's sign in conjunction with any one of the others is sufficient to establish a diagnosis, especially with a history of early syphilis.

Several diseases present characteristics which may be mistaken for locomotor ataxia.

1. *Peripheral neuritis.*—In one group of cases of this disease ataxia is present, and a mistake may be avoided by attention to the following points. There is generally a history of the action of a poison preceding the attack, and in the great majority of instances this is alcohol in some form, alone or in combination with arsenic ; lead or diphtheria may be the cause ; symmetrical paralysis and wasting, with altered electrical reaction of muscles, and the characteristic steppage gait are generally present ; but ataxia may be the predominating feature, paralysis and wasting not being evident. In these latter cases the difficulty is sometimes great ; but the history of the case assists, for in tabes a long time expires between the

beginning of the symptoms and the establishment of ataxia ; in multiple neuritis or alcoholic pseudo-tabes, as it is called, the advent of ataxia when it does occur is much earlier. There is marked muscular tenderness, and the pain which is present is not of the 'lightning' character ; the electrical reaction is altered, and though the knee-jerk may be absent the pupils are normal in their reaction to light. Moreover, the arms are frequently affected, which is the exception in tabes. *Diphtheritic paralysis* is excluded by the history ; and, though the knee-jerks are absent, the loss of accommodation and the paralysis of the palate are distinctive.

Cerebellar tumour gives a more or less characteristic gait of a swaying, drunken type, but the knee-jerk is often exaggerated ; the pupils are normal ; there is severe optic neuritis and occipital headache and absence of all the sensory and trophic phenomena of tabes.

General paralysis of the insane in its early spinal type presents a group of symptoms which may be practically identical with tabes, and only by a careful study of the mental condition can the case be properly catalogued. The advent of intellectual and moral degeneration which is a certainty in this affection lifts the curtain and reveals the true nature of the malady, which steadily progresses towards paralytic dementia. The onset of tremor in the tongue, lips, and hands, with difficulty in articulation, in a case supposed to be tabes should cause the physician to make an exhaustive investigation into the condition of the patient's memory, habits, and domestic behaviour, when, if these be found unsatisfactory, the case must be regarded in the far more serious light of general paralysis. It should be remembered that a syphilitic history is common in both diseases. The close association of the two conditions will be discussed under the heading of 'Paralysis, General, of the Insane.'

Disseminated cerebro-spinal sclerosis occurs at an earlier age, and may lead to signs of incoördination which have been mistaken for tabes, but the knee-jerks on examination will usually be found to be exaggerated ; the pupils re-act normally to light ; nystagmus, scanning speech, and the characteristic tremor will be present.

Paraplegia from whatever cause can only be confounded with the later stage of tabes, and the history is generally enough for differentiation. In ordinary paraplegia loss of power is the obvious sign, and the knee-jerk is exaggerated. In *Gowers' ataxic paraplegia* the knee-jerk is exaggerated and there is ankle-clonus, the lateral columns being always involved, and a spastic condition of the paralysed limbs supervenes.

Friedreich's ataxia has a marked hereditary history and occurs at an early age in children. The incoördination soon

affects the upper extremities and the head, and is of the cerebellar type. The knee-jerk is absent, but there are no eye or bladder symptoms, no crises or trophic changes. Nystagmus and hesitating speech are present.

Syringomyelia presents trophic joint and other changes resembling those found in tabes; the legs are weak and wasted and ataxia may supervene. The symptoms begin in the arms, and there is a remarkable loss of sensation to temperature and pain, whilst touch and muscle-sensation remain normal.

Prognosis.—Tabes has little effect upon the duration of life; the formidable crises are singularly free from danger, though it is possible death may occur in the laryngeal form. The disease, however, may be regarded as always progressive, if we exclude the pre-ataxic form, in which stage it may remain arrested for years. Considerable improvement may be manifested even in the second stage of the disease, and this may last for long periods. Forecasting the rate of progress is a difficult and often impossible process, and it is well to be satisfied with being wise after the event. As already noticed, the early optic atrophy is indicative of arrest of the spinal mischief, though the prognosis as regards sight is very grave.

Death, when it occurs, is owing to bladder and secondary renal changes, bedsores, or some intercurrent disease.

The writer recalls two cases which passed under his notice: one in which twenty-five years after the onset of lightning-pains the patient still continued to manage a large business, and though the sexual power had been more or less in abeyance for nearly twenty years it unexpectedly returned, and he became a parent. The other case ran through all its stages and ended in death within a period of six months.

Though the symptoms may be relieved by judicious treatment, and life may be prolonged, the disease itself must be regarded as incurable.

Treatment.—In those extremely rare cases where the disease has supervened at an early period after the syphilitic invasion, mercury and iodides have in a few instances given marked results, but as a rule ordinary antisyphilitic treatment is worse than useless. The chloride of gold and sodium salt seems to the writer to possess some power of retarding the degenerative process, and arsenic is believed to act in a similar mysterious manner; and upon the whole the best routine drug-treatment will be alternate monthly courses of each. The gold may be given in pills containing each one-tenth of a grain, and the arsenic in doses of half this amount in solution, three times a day. Nitrate of silver is recommended by several authorities; it probably has similar action, but it can only be given for short periods, owing to the danger of its staining the patient's skin.

Everything which improves the patient's general condition must be closely seen to, and gentle occupation of mind and body is desirable, but overwork, and over-fatigue in the pursuit of pleasure, and excesses of all kinds must be forbidden; exposure to cold and mental fagging certainly increase the severity of the lightning-pains.

The bladder-symptoms are of first importance, and life in many instances depends upon the conscientious way in which the catheter is kept aseptic; this may be further assisted by the use of creosote, salol, boric acid, or urotropin, internally, with the view of preventing decomposition of the urine in the bladder.

For the relief of the lightning-pains, rest in bed, the hot bath, morphia hypodermically, when *very* severe, and when of moderate intensity antipyrin and antifebrin, are all that is necessary. The more severe gastric and laryngeal crises yield to morphia, and sometimes to nitrite of amyl, which also sometimes relieves the pain; but in laryngeal spasm chloroform may be called upon.

Suspension, nerve-stretching, forced flexion of the spine, counter-irritation, and various vaunted electric cures, are now discredited; electricity is, however, of considerable value for the relief of certain symptoms, and the details of the different ways in which it may be employed will be found in the author's 'Dictionary of Treatment,' pp. 522–527.

The method of teaching the ataxic patient to perform various voluntary movements and combinations of movements, introduced by Frenkel, is of undoubted value; and when the services of a trained instructor cannot be obtained, much may be done by the patient himself, who can re-educate his muscles by the routine performance of exercises guided by his own direct vision and by a large mirror placed in front of him.

ATAXIA, HEREDITARY.

(See under FRIEDREICH'S ATAXIA.)

ATHEROMA. (See under ARTERIO-CAPILLARY FIBROSIS.)

ATHETOSIS. (See under HEMIPLEGIA.)

ATELEIOSIS

Is the name given by Hastings Gilford to a type of infantilism associated with dwarfage. The disease may begin in fœtal life, or after birth, as in the dwarf known as 'Tom Thumb' and many other exhibited cases. The mental development

may be good and the proportions of the body perfect ; the sexual organs are often stunted in development. The disease must be differentiated from cretinism by the mental brightness and the absence of myxœdematous signs, and from rickets and achondroplasia by the absence of deformity. Its pathology is unknown. (See under ' Infantilism.')

AUDITORY-NERVE DISEASE

Deafness and auditory disturbances are more commonly the result of diseases of the ear than of the auditory nerve, or of the centre for hearing, situated in the temporo-sphenoidal convolution. Ear-diseases belong to the province of the aural surgeon and need not be described ; only *nervous* deafness is here discussed. Primary inflammation of the auditory nerve, if it ever occurs, is a rare affection. Its primary degeneration is not uncommonly observed in tabes and disseminated sclerosis. Involvement of its peripheral portions may occur in the labyrinth from the extension of inflammatory processes in the middle ear. These latter, however, cause deafness of a mechanical nature from diminished conducting power.

The trunk of the nerve at the base of the brain may be affected by the pressure of tumours and aneurisms, by disease of the bones, and by meningeal inflammation, more especially by the basilar or cerebro-spinal form, which is liable to cause permanent deafness in infancy, and deaf-mutism.

Disease of the auditory nerve may show itself by loss of function causing more or less complete deafness, by subjective phenomena as tinnitus, in which buzzing and other distressing sounds are heard by the patient, and by hyperæsthesia (hyperacusis), in which case sounds are heard with abnormal distinctness. When that portion of the nerve (the vestibular branch) which goes to supply the vestibule and the semicircular canals becomes affected, there is interference with the maintenance of the equilibrium, or a sensation of giddiness, &c., which may prevent the patient from standing. This latter symptom-complex, known as Auditory Vertigo or Menière's disease, will be described under the heading of ' Vertigo.'

The deafness, tinnitus, and hyperæsthesia may be the result of the implication of the hearing centre, of the trunk of the nerve, or of the periphery of its cochlear branch, which constitutes the chief portion of the organ of hearing in the labyrinth or internal ear.

Deafness is, however, much more frequently caused by disease of the nerve in the latter situation ; but it is difficult to differentiate the centric deafness caused by disease of the nuclei within the medulla in the floor of the fourth ventricle from supra-nuclear disease occurring in the internal capsule, or

corpora quadrigemina, or in the temporo-sphenoidal cortex. Labyrinthine deafness is usually associated with tinnitus and often with vertigo, and so may be the deafness caused by centric mischief or by pressure on the trunk of the nerve. The causes of labyrinthine deafness may be a nerve-affection following inflammations of the middle ear, such as occur in measles and other fevers, and septic conditions ; hæmorrhages, syphilis, and cerebro-spinal meningitis also are important etiological factors. Poisoning by drugs, like quinine and the salicylates, also affects the labyrinth, and this form of deafness also is not uncommon in hysteria and profound anæmia, and in some blood-conditions, as those met with in Bright's disease, diabetes, and leukæmia.

The disease is often bilateral ; total deafness has sometimes followed hæmorrhage into both labyrinths, or been caused by syphilitic meningitis affecting both temporo-sphenoidal lobes, and by a tumour involving the corpora quadrigemina and medulla. The portion of the auditory path involved may sometimes be determined by the associated symptoms ; thus facial paralysis is liable to accompany labyrinthine deafness caused by pressure on the auditory nerve at the base of the brain, or in the internal meatus. (This association, however, may be the result of diseased bone or of middle-ear affection.) The sixth nerve sometimes suffers when the auditory trunk is involved ; and in the centric cases there are usually other symptoms indicative of hæmorrhage, or of growths in the pons and medulla, or of hemiplegia with hemi-anæsthesia when the cerebral hemisphere is involved.

In the differentiation of deafness or difficulty in hearing caused by disease of the nerve from the mechanical deafness produced by diminished conducting power caused by obstruction of the meatus or of middle-ear disease, the tuning-fork should be used. In mechanical deafness the vibrations of the tuning-fork held in front of the meatus are not heard, but they are perceived when the instrument is placed over the temporal bone ; this shows that the nerve in the labyrinth is free and responds to the sound conducted through its osseous walls. When the nerve-function is decreased either in the labyrinth or from involvement of the acoustic trunk, auditory path, or centre, this bone-conduction is lost or diminished. Rinne's test is used to demonstrate slighter degrees of impairment in hearing. When the vibrations have ceased to be perceived while the tuning-fork is kept in contact with the bone, in the normal state, they should be still audible if the instrument be held opposite the open meatus ; if mechanical deafness be present they are not perceived, but in minor degrees of nervous deafness the vibrations should be still audible. A ready, but not so reliable, test is that of Weber, who places the foot of the vibrating fork

on the centre of the forehead ; in mechanical or middle-ear deafness the vibrations are heard loudest in the deaf ear, but in deafness of nerve origin the vibrations of the instrument are only perceived in the sound ear. Nerve difficulty of hearing is also not so marked when tested by the patient's power of hearing ordinary conversation ; he perceives the sounds more distinctly than those of a watch in ordinary testing by this latter means, especially in a quiet room. Moreover, after the use of Politzer's bag the hearing is generally still further impaired. By closing the meatus bone-conduction is intensified, which shows that the difficulty in hearing is not in the nerve ; the intensification is the result of converting the meatus into a resonant chamber. In the aged, bone-conduction is often greatly diminished or absent.

Tinnitus, as already stated, is due to irritation of the auditory nerve ; it may follow any disease of the ear, it may even be caused by wax in the meatus, and it is very commonly present in nerve-deafness, whether this be centric or labyrinthine. It is sometimes functional, as proved by its presence in the epileptic aura, and in some cases of migraine. All sorts of sensations, from the faintest murmurs to the loudest whistlings or roarings, are experienced. In the centric cases the sounds are likely to be of the continuous type ; but such may occur in labyrinthine cases, though these are more likely to be intermitting and varying greatly in their intensity. They are heard whether the deafness accompanying them be unilateral or double ; they may be felt in one or both ears ; and sometimes they are perceived, not as if occurring in the ears, but as if they were produced somewhere else within the cranium.

They may be intense and troublesome when the hearing is apparently quite normal, and they have been noticed even when there was hyperacusis or increased sensitiveness of hearing, which, moreover, is present sometimes during the height of the attack of tinnitus, though more frequently the patient feels quite deaf when the sounds are loudest.

Tinnitus usually becomes less intense as hearing gradually becomes impaired and ends in complete deafness ; but it sometimes remains in those who become totally deaf. In one case under the writer's observation the patient subjectively heard several complete airs played upon a barrel-organ at different hours of the day. This patient's hearing was almost normal, and there was little doubt that the symptom was of centric origin.

The *diagnosis* of the cause is to be made upon the recognition of the associated symptoms, as those of disease of the middle ear or of some portion of the auditory tract producing signs of cerebral disease. When the sounds are of a pulsatile type aneurism may be present within the cranium, in which case the bruit may be heard by the physician through a stethoscope

applied to the cranium. Sounds caused by spasm of the stapedius and tensor tympani muscles may be accompanied by contractions of the facial muscles; and the clicking sound caused by the action of muscles connected with the Eustachian tube, according to Gowers, may be heard sometimes by another person at the distance of several feet from the patient's body.

Auditory hyperæsthesia is more commonly met with in disease of the nerve than in diseases of the ear. It is often functional, being not unfrequently present in hysteria. More or less hyperæsthesia, or *hyperacusis*, as it is called, is commonly observed in acute organic diseases of the brain, and especially in meningitis. In this latter condition the sounds are often, however, not intensified, but they produce pain or marked discomfort, always increasing the headache which is present, in which case the disturbance of the hearing function is styled *dysæsthesia acustica* or *dysacusis*. When the sound is appreciated by the patient as of a deeper or higher tone than it really is, the condition is known as *paracusis*.

The *treatment* of nervous deafness can only be met by the exhibition of remedies for the alleviation or removal of the morbid conditions in the brain which cause the difficulty in hearing; and as these conditions are usually beyond the reach of drugs, treatment is proportionately unsatisfactory. In early syphilitic cases much may be expected from mercury; in late cases the hope of benefit is for the most part futile, but large doses of iodides may be tried.

Tinnitus and hyperæsthesia are likewise to be met when possible by the removal of the morbid processes upon whose presence they depend; but even when this cannot be effected very considerable improvement and relief can be afforded by the routine administration of large doses of bromides alone or with small quantities of the newer analgesics—antipyrin, phenacetin, &c., combined with counter-irritation treatment.

BALDNESS. (See ALOPECIA.)

BANTI'S DISEASE.

(See ANÆMIA, SPLENIC; and also under SPLEEN.)

BERI-BERI

Is an endemic disease, probably of bacterial origin, characterised by the presence of multiple peripheral neuritis associated with more or less anasarca.

Etiology and Pathology.—The affection has been known in the East from the earliest times, and it is common in Japan,

Malay, China, South America, Central Africa, and in many other sub-tropical and tropical countries. It has been imported often into temperate regions, where it has remained endemic for considerable periods. Epidemics have frequently broken out in gaols, asylums, and barracks, and of late years the literature of these outbreaks in all parts of the world has become voluminous. The most interesting of these, because the nearest to home, was the prolonged epidemic in the Richmond Asylum, at Dublin, less than ten years ago ; but upon the whole it is rare in Europe, except when appearing as a ship-disease, and Europeans in affected districts always suffer less because their hygienic surroundings and food are generally superior to that of the natives.

H. Wright maintains that the disease is caused by a specific bacillus which he has isolated ; this causes acute toxæmia of the neurons, which reaches its height in three to four weeks and is liable to be followed by the true degenerative changes, which explain the residual paralysis.

Under the title of *Micrococcus berbericus*, Tsuzuki has recently described a diplococcus which he has isolated from the urine. It stains by Gram's method, grows on agar, is non-motile, ferments glucose and lactose and coagulates milk, and almost invariably gives a positive reaction with the serum of beri-beri blood. He has, moreover, proven the specific nature of this diplococcus by causing cardiac and nerve paralysis in animals by the injection of its soluble toxin and endotoxin.

Whether the causation of the disease be a microbe in the blood, or whether it be some chemical poison, the result of microbic activity outside the body, in the soil, or in food, there are several well-recognised predisposing causes. Overcrowding is the most important of these causes, especially in a moist and warm atmosphere. Though the disease is supposed not to be contagious in the ordinary sense (as are the exanthemata), there is absolutely no doubt about the virus which causes it being transported from one locality to another, where, under the favourable conditions just mentioned, it increases, multiplies, and continues to flourish for long or indefinite periods. The theory that it is a form of malaria, scurvy, or anæmia must be abandoned. It is, however, not so easy to eliminate dietetic causes in a study of the etiology of the disease. These probably act as predisposing causes like overcrowding, but food may be the medium, in some cases, for the conveyance of the specific germ or poison. Rice and raw fish were long maintained to be the cause of beri-beri, and this theory still survives in Japan ; and wonderful results have been obtained by a change of diet, in places where the disease is both endemic and epidemic. The use of fresh unshelled rice, when substituted for deteriorated grain

whose pericarp had been long removed, has checked the spread of the disease. Certainly the use of nitrogenous food, fat, and fresh vegetables, are potent factors in arresting an epidemic. Young males are most liable to become affected.

Symptoms.—The period of incubation certainly may extend to months, after which in many cases premonitory symptoms of malaise, feebleness in the limbs on slight exertion, with some pain, cramp, or transient numbness, may be observed for several days before the characteristic features of the disease begin to appear. These are the symptoms of a severe peripheral neuritis which may come on with striking suddenness in a single day. There is marked weakness in the legs and arms, with tingling and burning sensations, and numbness in the feet and hands. The calf-muscles are stiff, and become the seat of painful cramp, and are very tender when grasped. Œdema of the limbs and face appears, sometimes with startling suddenness, and tends to become general; and breathlessness with palpitation supervenes. The patient is soon unable to stand; his arms and hands become weaker, the muscular hyperæsthesia and cutaneous anæsthesia deepen, and the cardiac distress becomes more intense, and associated with it is pain in the region of the epigastrium. These symptoms, though they do not disappear, are not quite continuous, and between the exacerbations considerable relief occurs. The knee-jerks are soon lost, the urine becomes very scanty, there is no fever, and the mental state is unclouded. The patient may remain in this condition for several weeks, when the œdema begins to pass away with the advent of renal activity, as shown by marked increase in the urine; his other symptoms gradually improve, though now wasting of the muscles may be noticed, and this may be both general and very severe. When dropsy is slight the wasting may be obvious at the commencement. The duration of the attack is very variable and it may last many months, and relapses or recurrences are common.

The disease varies in intensity in each epidemic, and many types of it are described according to the predominance of certain of the symptoms. Thus in the so-called *dry* form there may be no œdema save a little pitting over the tibia, but the paralysis and muscular wasting may be so extreme in a few weeks that the patient presents the appearance of a skeleton, as if wasted by years. In mild cases of the same type there may be only weakness and slight wasting confined to the tibial muscles, with numbness of the fingers and toes; and these may not prevent the patient performing his ordinary work. In the *wet* form of the disease anasarca may be as prominent as in a severe example of acute Bright's disease, and effusion into the serous cavities may cause fatal embarrassment of the respiration and heart. In the *pernicious* type rapid paralysis of the diaphragm, intercostals, or cardiac muscles

may terminate life before the second day of the onset, the patient sinking from cardiac failure hastened by respiratory difficulties.

There are all forms of these types seen mixed up together, and severe and alarming symptoms may suddenly supervene upon any apparently mild attack, owing to involvement of the phrenics or pneumogastrics; or cardiac paralysis may bring on dyspnœa, cyanosis, or a fatal syncope at any stage of the disease, from dilatation of the right ventricle. The mortality varies exceedingly: in some districts it may be only 2 or 3 per cent.; in others, where the victims continue to remain in the affected locality and under the same evil hygienic surroundings, it reaches the appalling figure of 50 per cent. or more.

Morbid Anatomy.—Notwithstanding the variations in degree, type, and severity of the disease, the underlying pathological condition does not vary in its nature; it is a peripheral neuritis, and the pathological anatomy shows the destruction of the axis-cylinders and medullary sheaths of the nerve-fibres and degeneration of the muscles, in the main similar to or identical with the changes observed in alcoholic and diphtheritic neuritis. The cardiac muscle is always found affected in beri-beri, and often there is marked dilatation with fatty degeneration. Effusion into the serous cavities and into the cellular tissue and lung-substance is common; the nerve-changes may be also found in most of the special nerves, especially in the branches and plexuses of the pneumogastrics and in the phrenics.

Diagnosis.—In the epidemic form there is little difficulty; doubt generally arises in those isolated cases coming from the tropics, where malaria is common. The widespread peripheral neuritis, with dropsy and heart-complication, is never seen in malaria; but it must not be forgotten that both diseases may co-exist in the same patient.

From *alcoholic neuritis* the diagnosis is made by the history of the case. Evidence of continuous drinking is generally easily obtained, and other distinctive signs and symptoms of chronic alcoholism will be forthcoming. The tremulous tongue and characteristic shaking or tremor in the hands; the gastric condition, often with hepatic enlargement or cirrhosis; and the peculiar mental condition, usually easily differentiate the alcoholic affection. It is hardly necessary to repeat that the peripheral pain, cramps, hyperæsthesia or tenderness, and wasting of muscles, with the reaction of degeneration, anæsthesia of the skin, paralysis, and the well-marked high-stepping or shuffling gait, with drop-foot and drop-wrist, and the absence (generally) of the knee-jerks, are practically identical in all forms of multiple neuritis, whether produced by beri-beri, alcohol, diphtheria, or arsenic. The affection of the phrenic

and pneumogastric nerves in beri-beri, with the marked tendency to dropsy and cardiac degeneration, is of great importance in diagnosis.

Locomotor ataxia is eliminated by the tenderness of the calf-muscles, the drop-foot gait, the absence of the Argyll-Robertson pupil, and of the trophic and visceral disturbances, though the beri-beri victim may be unable to stand with his eyes closed.

Trichinosis may resemble beri-beri; but the tenderness and pain in the muscles are not associated with paralysis, and it chiefly affects the muscles of the trunk and neck; there is fever, and the affection is ushered in with severe gastro-enteritis.

Bright's disease is at once excluded by the absence of albuminuria. The suddenness with which the œdema of beri-beri comes and goes is often a highly characteristic feature of the disease.

Prognosis.—As already mentioned, the mortality is high, and the outlook very grave, in those cases not immediately removed from the affected district. The affection of the phrenic and pneumogastric, and cardiac degeneration, are serious. In some localities the prognosis is always grave, especially in very hot climates, where the wet form is common. Imported cases as a rule are not severe, but recovery of the atrophied muscles may be very slow, and weakness may remain for many months.

Treatment.—There is no special drug of value in this disorder, but the symptoms will indicate the choice of many agents which are beneficial. Thus, cardiac dilatation must be promptly met by strychnine hypodermically with nitroglycerin or nitrite of amyl, or even venesection in desperate cases. Removal to a higher altitude is essential in every case, or from the district where the disease has been contracted. Diet is of primary importance, and it should be liberal and not too bulky. Where rice has been the staple food, this should, if possible, be discontinued, and other cereals and animal food, with fresh vegetables and bread, substituted.

In mild cases the patient may be permitted to move about, but all severe cases and all cases in the active stage must be confined to bed, because of the danger of cardiac complications.

BILE-DUCTS, DISEASES OF.

(See under GALL-BLADDER.)

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BILHARZIA HÆMATOBIA

The disease produced by the presence of this parasite is known as Bilharziosis and sometimes as Hæmic Distomiasis.

Etiology.—This worm-shaped trematode, or fluke, is very common in Egypt and South Africa, where it is the cause of endemic hæmaturia and other troubles. The female, which is smooth, measures three-quarters of an inch (2 cm.) in length the male being smaller and tuberculated. The embryo finds its way into the body through drinking-water or by the eating of some freshwater fish or molluscs, which are supposed to be intermediary hosts of the parasite. The fully developed fluke resides in the veins of the portal system, in the renal, bladder, and rectal veins, and sets up severe inflammatory changes in the mucous membrane by its own presence and that of the numerous ova which escape from the veins into the surrounding tissues.

The ova are to be found in the urine; they are of the shape of melon-seeds, measuring ·12 mm. ($\frac{1}{200}$ inch), with a spinous process projecting from one pole of the ovoid; when the ovum is obtained from the rectum this spine is lateral, but Manson believes this characteristic belongs to a different species.

Much interest is attached to the recent discovery of another small fluke in Japan, by Katsurada, and named *Schistosomum Japonicum*, which produced an endemic disease allied to Egyptian bilharziosis. This parasite differs from *Bilharzia hæmatobia* (*Schistosomum hæmatobium*) by the absence of the papillæ or warts on the body of the male, and by the absence of the terminal or lateral spine on its eggs. The parasite is found in the portal and mesenteric veins, and eggs in the intestinal walls, gall-bladder, liver and brain, and other regions, but as yet its eggs have not been demonstrated in the bladder.

Symptoms.—The commonest symptoms produced by *B. hæmatobia* are those of bladder-irritation with hæmaturia, the act of micturition being painful and frequent, and followed by the passage of more or less blood, or by the passage of dark blood-stained urine. Sometimes the ureter or urethra gets blocked by the inflammatory thickening caused by the growth, and even pyelitis may supervene. Bladder-calculi are frequently due directly to the parasite. In the rectum polypoid growths resembling hæmorrhoids develop, which are the cause of tenesmus, pain, and hæmorrhage. Severe and often profound anæmia results, occasionally ending in death, and eosinophilia is constantly present. The liver-symptoms and signs may resemble those found in ordinary hepatic cirrhosis.

Symmers has found the ova in the pancreas and mesenteric glands and the mature fluke in the lungs, and has described the fibromatous changes in the liver causing cirrhosis, in

conjunction with the papillomatous growths in the large and small bowel, all in the same individual.

Diagnosis.—The presence of the ova, easily detected by a low power, is absolutely distinctive. The embryo is visible through the egg-case.

Prognosis.—This is generally good, especially in young subjects, who often spontaneously recover at puberty. But the parasite may remain in the body during the long lifetime of its host, who then is liable to continual or periodical attacks of bladder-trouble or calculi. The presence of renal disease, caused by the parasite, greatly increases the gravity of the case. When the lung and liver become involved, the prognosis is obviously graver.

Treatment.—Ordinary anthelmintics are useless, being unable to reach the parasites in the veins ; possibly the prolonged inhalation of chloroform might prove efficacious, but the writer is not aware that it has been tried. The symptoms indicate the remedies suitable for bladder-disease, and much relief is obtainable from systematic washing out of the bladder by boric-acid solution. Rectal growths should be removed, and agents like iron, arsenic, good food, &c., must be steadily administered, with the view of combating the anæmia.

The preventive treatment is obvious, and consists in the boiling and filtering of all drinking-water, and the prohibition of watercress and imperfectly cooked fish or freshwater molluscs. Bathing in the fresh water of the Transvaal, where the disease is very common, is believed to be the cause of the disease, the embryos finding their way into the urethra.

BLACKWATER FEVER

This is an acute specific fever characterised by the presence of hæmoglobinuria.

Etiology and Pathology.—This affection, known by at least a score of different names, is a common disease of tropical and sub-tropical Africa and America, hence its common name of West African Fever. It is often described as Bilious Remittent or Hæmoglobinuric Fever. The result of the great and sudden destruction of the red blood-corpuscles, probably caused by a protozoon, accounts for the hæmoglobinuria.

The most diverse opinions are still held about the pathology and cause of this affection. Most authorities maintain that it is a form of malaria, as the malarial parasite has been so often seen in the blood, and unquestionably nearly every patient suffering from the disease has also formerly suffered from malaria. It is, however, held by others that the presence of the malarial parasites is a coincidence, since the two diseases may occur together, and blackwater fever is always met with in

the malarial zone, especially in equatorial Africa. The recent figures of Stephens, quoted by Osler, show that the malarial parasites were found in over 95 per cent. of the ninety-five cases examined—a fact which seems positively conclusive as to the malarial origin of the disease, though the plasmodium may prove to be of a different species from those hitherto discovered.

The theory supported by Koch, that it is simply the result of quinine-poisoning, must be discredited, as the disease is met with in its typical form in natives who have never taken quinine; but this drug often will bring on an attack when the hæmoglobinuric condition is present.

Various parasites have been described, but the real causative agent has not been demonstrated yet. Sambon maintains that it is identical with the protozoon which produces the Texas redwater fever in cattle, and that blackwater fever is caused in the tropics by an introduction of this parasite into the blood of patients who have suffered previously from malaria.

Symptoms.—The invasion begins with a severe shivering fit, or a series of rigors accompanied by bilious vomiting, and the passage of dark, thick, viscid urine; the temperature reaches 103°–105°, and in a few hours jaundice appears, and this deepens to a rich lemon tint. The course of the fever is very irregular: it falls in four to six hours after the rigor, and may in a few hours rise again with another shivering fit. In other cases there is a remission of one or two days between the paroxysms, or these may follow each other rapidly till death, which may occur in seventy-two hours, but more often death does not occur till about the sixth day. Relapses are common, and the only two cases of the disease seen by the writer were examples of these where death occurred upon the seventh day, in one of them from total suppression of urine. Sometimes the intense vomiting produces a fatal collapse preceded by hiccough. In many cases the patient gets well spontaneously before the end of a week. After the drop in the temperature the urine rapidly improves in colour and in quantity. It is generally of high s.g., and acid, though of a dark red or black; few, if any, red cells are found, the colour being due to hæmoglobin; there is always much albumin (cell-globulin).

The blood from a finger-prick shows rapid and extensive destruction of red corpuscles, and marked leucocytosis. The serum is yellow, and pigmented leucocytes are also present. There is enlargement and tenderness of the spleen and liver. When different malarial parasites are present the symptoms may present a mixture of types.

The mortality varies from 60 in Central Africa to 15 per cent. in many sub-tropical regions.

Diagnosis.—*Yellow fever* has been mistaken for hæmoglobinuria. The fever is, however, regular, and relapses are rare. The vomiting is black, consisting of altered blood, and does

not consist of bile, as in hæmoglobinuria ; and the urine always contains serum-albumin, and only gets dark in the later stages from the presence of hæmorrhage. The jaundice appears later in the disease when it does appear, and never is intense at the early stage at which it is seen in hæmoglobinuria, and there is no marked splenic enlargement.

Malarial fever is maintained by those who still cling to the belief in its distinct identity from blackwater fever to be distinguishable by the greater regularity of the paroxysms, and the generally higher temperature. The urine in malaria may be dark from bile, and if kidney-complication arises, blood-corpuscles are obvious ; but it differs always from the viscid black liquid loaded with hæmoglobin which is absolutely characteristic of blackwater fever ; the jaundice in malaria is always slight, and never appears at the onset of the attack.

Prognosis.—This depends largely upon the locality, and the gravity of the case deepens with the number and severity of the paroxysms, and especially with the scanty volume of the urine.

Treatment.—This will consist in the relief of symptoms as they arise by the administration of recognised agents, as diuretics, cathartics, &c., and the application of poultices to the painful liver, with hydropathy and diaphoretics for the relief of urinary suppression. Large injections into the colon, or into the subcutaneous tissue, of warm saline solution are valuable in this latter condition.

Much has been said in condemnation of quinine, and still many affirm that if it be not the invariable cause it at all events always aggravates the disease. It must be acknowledged that it has no specific action in the acute stage of the hæmoglobinuria, and that it should not be administered except in those cases where the malarial parasite is demonstrated to be present in the blood. Most authorities, however, agree that the after-treatment of the disease must consist in very small and cautiously increased doses of quinine, and Stephens maintains that the cure cannot be considered complete till the patient has been enabled to bear 15 grs. without ill-effects.

BOILS

These are localised inflammations of the skin and subcutaneous tissue, originating in the sweat-glands or around the hair-follicles, caused by the presence of Staphylococcus pyogenes aureus or albus, and eventuating in circumscribed abscesses.

Pathology and Etiology.—The irritation caused by the pus-forming microbes leads to the formation of an acutely painful indurated swelling, surrounded by an angry red margin ; the thickened area increases in size, breaks down in

the centre, which is occupied by a characteristic slough, over which a small pustule forms, and discharges in three or four days. The underlying slough slowly separates and is discharged piecemeal, or as a firm greenish core, leaving a small abscess-cavity which heals by granulation. The favourite sites are the buttocks, nape, and axilla, but any part of the skin may be involved. The pain which boils produce depends upon the tension caused by the swelling; this is severe in the auditory meatus and in the nose.

Probably always there is some constitutional factor which diminishes the natural resistance of the tissues to the local invasion of the parasite; but the boil, or furuncle, as it is also called, must be regarded and treated as a local and not as a constitutional disease. The constitutional or predisposing factor is seen in the frequency with which boils are met with in diabetes and Bright's disease, and during recovery from typhoid and other fevers, and in drunkards.

Carbuncles, though differing apparently from furuncles in their greater dimensions, more extensive brawny indurations, the presence of several separate apertures leading down to the large central core, and their grave constitutional complications, are nevertheless the result of the same pathological process, and only differ in degree from the furuncle.

Treatment.—When the boil is seen at its commencement, the suppurative process may be aborted by plucking out the central hair at its summit, scraping the affected skin with a sharp scalpel, and painting over with lunar caustic, strong iodine solution, liquid carbolic acid, or other powerful antiseptic. If the affected area be large, as in carbuncle, a few drops of carbolic acid may be injected at several places. Herman has recently reported excellent results in the treatment of boils and carbuncles in their early stage by injecting through an ordinary hypodermic needle about a litre of oxygen under a pressure of 120 atmospheres into the tissues under the skin in the neighbourhood of the inflamed area.

When already severe pain, tension, and throbbing are felt as the case comes under observation, great relief is obtainable by poulticing. As soon as the process of suppuration has made itself evident, a free incision, followed by antiseptic poulticing or water-dressings gives immediate relief.

Of very great importance in all cases are the measures which tend to promote increased resistance of the tissues, as improved hygiene, &c., and the local cleansing and disinfecting of the skin. Auto-infection is the explanation of those cases of recurring boils which cause so much trouble; and as the skin in their vicinity must be regarded as infective, steps should be taken for the destruction of all pus-forming germs on the surface of the body, and also upon the clothing. Nothing is better than a daily warm bath, to which enough

permanganate of potassium has been added to cause faint staining of the skin ; stronger solutions or sublimate lotions being afterwards applied to the skin in the vicinity of the boils.

The general management of carbuncle will proceed upon similar lines, but here generous stimulation may be demanded to counteract the depressing effect of the absorption of the local poison. The best procedure is a free and deep crucial incision into the centre of the indurated mass, after which all sloughing tissue or core should be scraped out and the cavity swabbed freely by strong carbolic acid, and treated afterwards upon general antiseptic surgical principles. Excellent results have been obtained by Wright's vaccine treatment.

BRAIN DISEASES.

(See under CEREBRAL ABSCESS ; CEREBRAL HÆMORRHAGE ; MENINGITIS ; PARALYSIS, &c.)

BRIGHT'S DISEASE, ACUTE

This may for all clinical purposes be regarded as an acute non-suppurative nephritis or inflammation, involving the component parts of the renal organs—*i.e.* (1) tubules ; (2) glomeruli, and (3) interstitial tissue. These different structures are involved in different degrees, and hence the nomenclature, Acute Desquamative Nephritis, Acute Glomerulitis, Acute Interstitial Nephritis, or, more tersely, *acute tubal*, *glomerular*, and *diffuse* nephritis. It is safer in the present state of our knowledge to regard these as merely different varieties of one disease—nephritis ; the variations probably depending on the selective action of the morbific cause upon the renal constituents.

Etiology and Pathological Anatomy.—The causes are many, the most usual being (1) Exposure to cold and damp, which suddenly throws more work upon the kidney than it is able to accomplish ; (2) the circulation through the gland of the toxic products of scarlatina, diphtheria, and other fevers ; (3) the irritation resulting from the administration of such poisons as chlorate and nitrate of potassium, cantharides, turpentine, phenol, &c. ; (4) interference with the circulation in the renal artery or vein, as occurs in valvular lesions and pregnancy ; (5) interference with the function of the skin, as in extensive scalds, burns, and acute general dermatitis.

The naked-eye characters of the renal change, are in a severe typical case obvious ; the organ is increased in size and weight, and is markedly hyperæmic, the capsule being thin, smooth, and non-adherent. On section the cortical part is seen to

make up most of the increased bulk of the organ; it shows the congested glomeruli as intense red specks. The microscope will reveal the tubules filled with shed epithelium, blood-cells, fibrin, casts, and granular *debris*; the glomerular tufts are compressed by inflammatory cell-products poured out inside the Malpighian capsule, which is thickened, and the capillaries are often seen undergoing hyaline degeneration; there is new fibroid and nuclear proliferation of the scanty intertubular or interstitial tissue.

The glomerular changes are always well marked in scarlatinal cases, and may be advanced before the other structures are much involved. At a later stage of the disease the swollen cortical part of the organ assumes a mottled appearance, or often is markedly pale in colour, contrasting with the congestion of the radiating Malpighian pyramids.

The disease is most common in young subjects and in males.

Symptoms.—The first symptom may be dropsy, especially in exposure cases; this generally is noticed as puffiness about the eyes, face, and hands, rapidly becoming general, though it may remain slight. With it, changes in the urine are observable: the secretion is diminished, though micturition is often frequent; generally it shows unmistakable evidence of containing blood, and may resemble porter in appearance; it has a high s.g., which may register over 1030 in some cases. Always it contains albumin, and this may be so abundant that the liquid becomes quite solid on heating. The amount of albumin in every case is much greater than the blood which is present will account for. A dark-coloured abundant sediment falls down in the urine-glass on standing, and this, under the microscope, shows numerous red corpuscles in various stages of disintegration, and epithelial cells from the interior of the tubules and epithelial *debris* and casts. In the cylindruria of acute Bright's disease samples of all the kinds of casts may be met with in the same urine; but the characteristic forms associated with acute nephritis are the epithelial and blood casts. They are of different sizes, according to the diameter of the tube in whose lumen the cylindrical body was formed. If the cast be formed in a tubule whose epithelium has been shed, it will be large in diameter.

The above signs of œdema and urinary change may follow a few hours after exposure to cold, or they may not be noticed till the patient gets up on the following morning. There may be slight or marked fever, vomiting, malaise, chills, lumbar pain, and headache. The onset may be insidious; but as a rule it is definite, and sometimes it may come on with explosive suddenness. The writer once saw a scarlatinal patient who reported himself as feeling quite well and convalescing without a complication, when a uræmic convulsion

occurred within a few minutes—the first indication of an acute nephritis which had been slumbering.

The œdema increases generally with the diminution of urine, and the anasarca may become so extensive as to threaten life from accumulations in the serous cavities, lung, or laryngeal tissue. The impure blood circulating through the vessels may become the cause of fatal pericarditis or pleurisy. The pulse may be slow or quickened, but it rapidly becomes hard and not easily obliterated by the pressure of the finger; whilst the cardiac sounds are modified, especially the accentuation of the second sound to the right of the sternum, showing greatly increased arterial tension, under which the embarrassed heart may suddenly dilate. Reduplication of the first and of the second sound is often observable, but as a rule the short duration of the disease does not admit of marked hypertrophic changes occurring in the ventricles.

Uræmic symptoms are not uncommon, and their occurrence depends upon the amount of the diminution of the urine and the acuteness of the onset; the patient's nerve-centres not being accustomed to the uræmic poison, as in the chronic forms of renal disease, a comparatively slight charging of the blood with the specific poison will sometimes determine a convulsion (see under 'Uræmia'). When suppression occurs, the danger is great, and uræmic symptoms may come on before the appearance of dropsy. The headache is usually intense in these cases, and there may be dull pain over the kidney-region, which is sometimes slightly tender on deep pressure.

The course and duration of the disease vary greatly: in very mild cases recovery may be complete in three or four weeks, whilst in other cases it slowly drifts imperceptibly into the chronic form of Bright's disease. When a period of six months' duration has expired without recovery, for all practical purposes the disease may be regarded as chronic.

The complications seen in acute Bright's disease are numerous; whilst hydrothorax, œdema of the larynx and lungs, &c., may be regarded as belonging to the symptomatology of the affection, as are also uræmic convulsions, often true inflammatory complications manifest themselves, as acute pleuritis, pericarditis, peritonitis, and lobar pneumonia. Hæmorrhages are not uncommon, and epistaxis may be copious; retinal hæmorrhages may cause failure in sight, which is to be differentiated from the more fleeting amaurosis caused by the uræmic condition.

Diagnosis.—This is seldom so easy in practice as a perusal of the literature of the disease would usually suggest. True it is that the sudden uræmic symptoms—dropsy, and the characteristic dark-coloured, highly albuminous and scanty urine, soon following after a chill or occurring during convalescence in scarlatina or other disease—form a well-marked clinical

picture; but the question must always arise, Is this acute nephritis, or is it an acute attack supervening upon a *chronic form of the disease*? Upon the answer the prognosis largely depends. If it be known that the patient's urine was free from albumin a short time previously, the case can scarcely present any difficulty. A considerable percentage of the patients seeking admission to hospital for kidney-disease do so because they have noticed blood in their urine for the first time, and this has appeared after a severe wetting; a careful examination of the history of these cases often affords unmistakable evidence of previous disease. Thus dropsy may have been present, though slight in degree, and symptoms of chronic uræmia affecting the special nerves of sight and hearing may be elicited. In these doubtful cases a retinal examination may be of great value, as old scars left from previous hæmorrhages may be demonstrated by the ophthalmoscope. The microscope in skilled hands will often reveal signs of chronic degenerative changes, fatty and hyaline, in the epithelial elements, which may be so extensive as to justify the opinion of long-standing disease; but this is a question of great nicety, requiring much experience, and Emerson maintains that it is impossible without a study of the clinical history. In a few cases time alone will settle the diagnosis. These remarks also apply to the albuminuria occurring in pregnancy.

The febrile albuminuria constantly occurring in the early stages of pneumonia, scarlatina, erysipelas, and other fevers must not be confounded with acute nephritis. It is always slight in amount, fluctuates with the alternations of the fever and disappears as the temperature falls. It must therefore be carefully differentiated from the serious form of albuminuria indicative of acute nephritis, which comes on at a later stage of these diseases during convalescence, though in rare cases the two forms may run into each other. The supervention of nephritis is so common in scarlatina that repeated examination of the urine should be a matter of routine during the desquamating period, and for a month afterwards.

In *diphtheria* the albuminuria should be regarded as part of the disease, and rarely as a sequela. It is seldom serious; and though more frequently present, and lasting longer than the ordinary febrile form, it is but rarely the cause of acute nephritis. When this does supervene, generally it is of the simple tubular form, and never shows itself as the formidable glomerulitis which is constantly seen following scarlatina.

Infantile forms of acute nephritis are said to exist with dropsy, but without albuminuria, but such must be very rare.

Hæmorrhages from the pelvis of the kidney, ureters, or bladder may produce such a changed condition of the urine as to lead to mistakes; the albumin in these cases is proportionate

to the amount of blood ; casts in great numbers are not present, and dropsy is absent.

Hæmoglobinuria is characterised by the red coloration ; but even in cases where the tint may be dark-brown, there are no red blood-corpuscles, but albumin is always present. In *blackwater fever* this test is also conclusive.

A diagnosis made upon the presence of casts alone is apt to be fallacious ; even blood-casts are found often in the urine of athletes in company with hyaline casts after severe muscular effort, though the kidney may be intact. A *pure* cylindruria, in which casts may be abundant, has been noticed under similar conditions without albuminuria, and is said to occur after the administration of salicylates and other drugs, and it is sometimes found in cases of the small white chronic kidney.

Prognosis.—This deepens with the length of the duration ; but it is upon the whole favourable, unless in the scarlatinal form in very young children, when it is often fatal. Prognosis in cases directly caused by exposure to cold is nearly always favourable. The presence of uræmic symptoms, even of a formidable type, need not cause great alarm if treatment be prompt and judicious.

The danger must be always recognised of the case passing into the chronic type ; but recoveries are not rare where the disease had lasted from six to nine months.

The prognosis is also largely influenced by the complications of the condition to which the nephritis is secondary, as in typhoid fever, pregnancy, scarlatinal otitis, &c. It must be noticed that the acute nephritis of pregnancy is, especially in subsequent repetitions of the pregnant state, very liable to take on the chronic form and end in the small red kidney.

Treatment.—The patient should be at once placed in bed, clad in light flannels and enveloped in blankets, linen sheets being dispensed with. His diet should consist of milk, gruel, barley-water, and diluent drinks, animal food and solids being forbidden. After the subsidence of the acute symptoms, farinaceous foods and soups may be given. Alcohol in every form must be avoided.

The function of the kidney being in abeyance, the treatment must be directed to the skin and bowels, with the view to their more active elimination of water and poisonous waste-products. Sweating is to be encouraged in every way by simple diaphoretic drugs like Mindererus spirit and spirit of nitre ; warm, hot-air, and vapour baths are also indicated with the view of assisting these. In the presence of uræmic symptoms the quickest diaphoretic will be the hot pack, alone or with mustard, and after the skin has commenced to act, *and not till then*, a hypodermic of pilocarpine nitrate

$\frac{1}{3}$ gr. may be given. Copious drinking of whey and acidulous drinks or weak barley-water should be permitted, even when the dropsy is extensive.

Purgatives of the saline class—the most reliable of which is magnesium sulphate—should be freely given with the view of evacuating large quantities of fluid by the bowel. With this drug alone uræmic symptoms may be fought successfully and extensive dropsies removed, especially if it be administered in fairly concentrated form.

Diuretics are very uncertain, and in some cases dangerous, and the routine administration of irritating or stimulating drugs like juniper, squill, digitalis, or gin is highly objectionable. Water is the only absolutely safe diuretic in severe cases. When arterial tension is very high, nitrites are invaluable from their action in reducing the renal blood-pressure, and the spirit of nitrous ether never does harm. Digitalis is only indicated in those rare and serious cases where the blood-pressure is low. When uræmic convulsions do not yield to the above measures, a large vein should be opened and ten to twenty ounces of blood removed, and three or four pints of saline solution injected hypodermically into the loose cellular tissue of the flanks, axillæ, and groins. In puerperal cases this, in the writer's hands, has saved life after every other means had failed.

Other symptoms must be treated as they arise, vomiting being relieved by gastric sedatives, ice, and counter-irritants. Certain drugs should be avoided in the treatment of acute nephritis—*i.e.* cantharides internally or externally, calomel, morphia, and chlorate or nitrate of potassium.

Where localised dropsy is very great, aspiration of any of the serous cavities may be undertaken to relieve urgent symptoms, or it may be necessary to puncture the skin (under strict antiseptic precautions) with the view of relieving its great tension, or to insert a Southey's tube.

Pain in the loins may be relieved by dry-cupping, pain in the head by sinapisms at the back of the neck; thirst is eased by the popular potus imperialis, made by adding a large teaspoonful of cream of tartar to a quart of boiling water, to which a little sugar and lemon have been added; it may be taken iced. Kali-water and diluted lemon-juice in double effervescence is very grateful, and it thus supplies one of the least objectionable of all diuretics—the citrate of potassium.

After the subsidence of the more acute symptoms, when the dropsy is disappearing and the urine is increasing, iron is nearly always indicated to combat the anæmia; the tincture of the perchloride—20 mins. in four times as much liquor ammoniæ acetatis and a little water, is the best form for administration. No drug has any influence upon the

albuminuria, and the so-called astringents—ergot, lead, tannin, &c.—are injurious.

The return to animal food and solids must be gradual, and the greatest care must be exercised as regards exposure to cold and damp.

BRIGHT'S DISEASE, CHRONIC

Under this name are grouped several distinct affections. The group is still, unfortunately, not very clearly defined, as authorities differ as to whether some of the members are instances of different diseases, or of the same disease at different stages of its progress. The nomenclature is also unsatisfactory, several names being used in the designation of each member of the group: one name being indicative of the naked-eye characters of the diseased kidneys, another of its microscopic features, whilst a third title is used sometimes to embody a debatable theory of its causation.

The most commonly seen member of the group, and consequently the one of greatest clinical importance, will be described first; it is known by its macroscopic characters as the

LARGE WHITE KIDNEY

Other names are, *Chronic Tubal Nephritis, Chronic Desquamative Nephritis, Chronic Parenchymatous Nephritis.*[1]

Etiology.—This is the form of chronic Bright's disease which is accepted to be the result of acute nephritis, when this affection, after a period of four to six months, passes into the chronic stage. Under these circumstances, its causation or etiology will be that of the original condition which produced its acute stage—*i.e.* exposure to cold and damp, a morbid poison like that of scarlatina, or some obscure condition connected with the pregnant state. It more frequently, however, begins insidiously with all the characters of a chronic disease, and its causation may be said to be then unknown, dating back to some remote illness or condition not suspected. The long list of causes, such as alcoholism, heart-disease, lead and other mineral poisons, syphilis, malaria, gout, tubercle, overwork, heredity, &c., have little evidence to support them, though all these factors may exert a predisposing

[1] Much confusion results from the plan of classifying the types of chronic Bright's disease under the subdivisions, Chronic Tubal and Chronic Interstitial Nephritis. In chronic tubal nephritis there are always interstitial changes; but it is only when the shrinkage of the new interstitial tissue leads to secondary changes in the vascular and secretory apparatus that the term 'interstitial' should be employed.

influence. The disease is more common in males before middle life, but is to be met at all ages. It is more common amongst the ill-clad and ill-fed, and especially amongst those whose occupations expose them to constant wettings.

Morbid Anatomy.—This is implied in its name. The kidney is enlarged and may scale two or three times its normal weight; it is pale in colour and smooth on the surface, and its thin capsule readily strips off, showing stellate venules on its surface. On section the red pyramids contrast in colour with the pale and enlarged cortex. The microscope shows: (1) The dilated tubes distended with epithelial cells in varying stages of granular, fatty, or hyaline degeneration; and casts are present *in situ*; (2) the Malpighian capsules are thickened, the vessels in the capillary tufts are also thickened by cellular infiltration, and show hyaline change, and the epithelium over them is altered; (3) the interstitial or intertubular tissue is hypertrophied—*i.e.* there is evidence of new fibroid tissue and small-celled infiltration. (Sometimes hæmorrhages into the tubes and around them are visible, and then the kidney is classed as representing a variety of the disease called *chronic hæmorrhagic nephritis*.) Both kidneys are always involved, though the changes may be more advanced in one.

Accompanying the above condition of the renal organs, well-marked secondary lesions are found in the vascular system, which in the main correspond with those observed in arterio-sclerosis, and consist of thickening of the arterial coats, due to muscular hypertrophy, followed by degenerative changes of a pronounced type in all the coats. The heart, under the strain caused by the increased peripheral resistance, shows marked hypertrophy of its muscle, and at a later stage myo-cardial degeneration sets in, followed by dilatation and relative insufficiency. These changes, though often obvious, seldom proceed to the extent observable in the cirrhotic kidney. Occasionally the results of secondary inflammatory lesions may be observed in the serous membranes, and not infrequently the nature of the exudates is purulent and often mixed with blood.

Symptoms.—When the disease follows acute nephritis there is no practical difference in the symptoms, the diagnosis being made by the duration of the attack; and it is a good clinical rule to label the disease as a case of the large white kidney of nephritis when the symptoms of dropsy and albuminuria have lasted for six months after their first onset in an acute form.

Generally, however, the period of commencement is indefinable: a history of slight transient recurring anasarca of the legs, with pallor, loss of appetite and strength, is elicited; and when the urine is examined it is found to be characteristic of the established disease. Not infrequently the discovery

is made during the routine examination for life insurance of a subject who is unaware of any serious departure from health, or during the testing of the eyes for failing accommodation or errors in refraction.

The clinical picture in advanced cases of the disease can scarcely be mistaken. The patient is markedly pale though not emaciated, and there is some œdema or puffiness of the eyelids; these, together with undue dryness of the skin, give a pasty appearance which is characteristic. The anasarca is always a marked feature, and though at first it may be confined to dependent parts, it becomes universal, involving the genital organs, the serous cavities, the lungs, and even the conjunctivæ, producing a water-logged condition which affects the heart and breathing. Often the tension of the pulse is increased, the aortic second sound is accentuated, and there is evidence of hypertrophy of the heart. Reduplication of the cardiac sounds is not unusual, and at a later stage the signs of failing compensation may show themselves. Evidence of the effect of retained waste-products upon the nerves of special sense is always present, deafness, or tinnitus, and dimness of vision being often noticeable; and retinal hæmorrhages and intense headaches, with nausea, vomiting, or formidable diarrhœa, probably of an eliminatory character, are complained of. Hæmorrhages are not infrequent from the mucous surfaces, especially from the nasal membrane, and these tend to heighten the characteristic pallor which is always present. Uræmic dyspnœa, coma, and cerebral hæmorrhages are less common than in the cirrhotic form of the disease.

The character of the urine is of first importance. It is diminished in amount, sometimes by one-half, pale in colour, unless temporary exacerbations may lead to hæmorrhage, when the colour may be smoky or dark, as in the acute attack.

The s.g. is below the normal, but during active treatment by profuse sweating and purging the density may approach or exceed the normal, and it may then become cloudy on cooling, from the deposition of urates; but if the volume and density be considered together, there is *always* evidence of a diminution in the amount of urea excreted.

Albumin is abundant, sometimes to the extent of the deposit occupying more than one-half of the test-tube, and the daily amount excreted may exceed one ounce avoirdupois (28 grms.).

Upon standing, the sediment which falls, when examined, will show generally abundance of casts—epithelial, granular, fatty, hyaline, and sometimes bloody. The casts are of all sizes, some large ones being evidently formed in tubes whose epithelial cells have been already shed. Degeneration of the epithelial elements entering into the formation of the casts is generally obvious, and some idea of the widespread and

grave destruction of the secreting structure may be obtained by repeated microscopic examination. The presence of amyloid kidney is not to be inferred from the appearance of waxy or hyaline casts, for the amorphous type of cast may be found in almost every case.

Almost of equal importance are the amount and nature of the loose cellular elements. The polygonal nucleated renal epithelial cells often show extensive fatty degeneration ; leucocytes, red corpuscles, and tailed cells are also present, with much granular and fatty *debris*.

The symptoms and signs of the inflammatory complications, as pleuritis and pericarditis, differ in no way from those found in other affections, but the liability to a suppurative termination must be kept in mind. The disease is progressive and indefinitely chronic in its course, often lasting for very many years.

Diagnosis.—Generally there is little difficulty in coming to a conclusion upon this point, the only danger of confusion being in mistaking the disease under consideration for acute nephritis if the patient has recently been exposed to a severe chill or wetting (see p. 113).

In the large white kidney of chronic Bright's disease, the amount of albumin in the urine reaches a figure not to be met with in any other chronic renal condition ; this, with the abundance of anasarca and the presence of casts, is always characteristic. When the disease has lasted for a very long time a difficulty may, however, arise in the change which may occur in the volume of the urine ; this will be referred to under the heading of 'Small White Kidney,' on next page.

Amyloid disease is recognised by the history of its cause—*i.e.* syphilis, prolonged suppuration, or phthisis ; by the evidence of disease in other organs, as in the spleen and liver ; by the absence of dropsy, and the increased amount of urine.

The *small red kidney* is eliminated by its own marked characters—*i.e.* increased flow of slightly albuminous urine, absence of dropsy, and presence of marked vascular changes.

Valvular disease with much dropsy has its own history and signs, and the amount of albumin always is small when compared with that found in cases of the large white kidney.

Prognosis.—This is always most grave ; as a rule, in adults the disease is a fatal one. The gravity of the case deepens with the extent of its duration. In cases with a clear history of origin in an acute attack six months before, some slight hope may be indulged in ; but this hope must fade as months pass, and it should vanish completely by the end of a year, except in the case of children, where recovery sometimes follows after a still longer duration.

Life may be threatened at any period by uræmia, acute inflammation of a serous membrane, œdema of the lungs or

larynx; but these may be staved off by judicious management for two or three years, and in some cases for longer periods. The prognosis is always very grave where the total amount of urine steadily diminishes, when its s.g. is very low and there is an excessive amount of albumin, and when marked fatty casts, with fat-droplets, begin to appear in the urine.

Treatment.—This is to be carried out upon the same general lines as have been sketched under 'Acute Bright's Disease.' The details of treatment indicated by the different complications are discussed in the fourth edition of the author's 'Dictionary of Treatment,' pp. 83–92, and need not be dwelt upon at length here. The main principles of treatment should consist of an attempt to transfer the functions ordinarily performed by the renal organs to the skin and mucous membrane of the intestinal tract, and to meet the loss of albumin daily excreted. Warm clothing, baths of various kinds, or change to an equable warm climate, to ensure the constant and thorough over-action of the sweat-glands, and the continuous use of saline purges, are clearly indicated; as is also a diet which will throw the least possible work upon the diseased kidney. Anæmia must be met by iron, judiciously administered, and alcohol in every form forbidden.

The modern operation of decapsulating both kidneys has been carried out in numerous cases, sometimes with marked benefit to the anasarca and respiratory embarrassment; but the procedure cannot be recommended as a routine, and the mortality after it is so high that it seems likely to be abandoned. There is usually no evidence of any marked collateral circulation being established by the new capsule which speedily forms, and at best the operation can but temporarily relieve any increased renal tension present.

SMALL WHITE KIDNEY

This is a rare condition, and its existence is mainly of pathological interest; little clinical importance has been attached to it in the discussions which its histological characteristics have excited. It is also known as the Pale Granular Kidney.

Pathology.—It is the form of disease which is supposed to supervene upon the large white kidney in those cases where death has been long postponed under the conditions last described. Some observers believe that it takes its origin in acute nephritis (acute Bright's disease), which, after passing through the large white or chronic stage, winds up as the small white kidney. Others affirm that it may supervene upon those cases of large white kidney which were essentially chronic in their onset; whilst not a few regard it as simply a phase of the red granular or contracted kidney to be next described Rose Bradford holds the view, which is accepted as correct by F. T. Roberts, that the small white kidney is in some

instances a distinct pathological primary and independent entity in which the changes are of a degenerative character, differing altogether from those met with in the small red kidney.

The important and practical point, therefore, for the clinician to observe, is that at the bedside its symptomatology may be regarded as identical with that of the small red granular or contracted kidney of chronic interstitial nephritis, regardless of theories about its origin. Its presence may be suspected or speculated upon when polyuria and other signs of the small red kidney become grafted upon the symptoms of large white kidney in young adults or in patients who have not yet reached the middle period of life. Its pathological anatomy is roughly sketched in its name, but, unlike the large white kidney, its surface has lost its smooth outline, owing to the contraction of the new fibrous tissue which has been proliferated between the tubes. This, as it shrinks, causes puckering or indrawing, which leaves a dimpled or granulated surface and adherent thickened capsule. By this contraction, which is most marked on the surface, the tubular and vascular elements already extensively diseased are subjected to pressure which causes atrophy, and the gross result is that the organ may be diminished to half of its normal weight, producing a train of symptoms which in their diagnosis, prognosis, and treatment may be regarded as practically identical with those of the small red granular kidney.

SMALL RED KIDNEY

Here the complexities of nomenclature reach their limits, as this form of chronic Bright's disease is also recognised under the following names—viz. *Chronic Interstitial Nephritis, Renal Sclerosis, Cirrhosis of the Kidney, Contracted Kidney, Gouty Kidney, Lead Kidney, Red Granular Kidney, Arterio-sclerotic Kidney, Primary Contracted Kidney*, &c.

Etiology.—The causation of this disease is unknown in many instances. A very trifling percentage of cases is supposed to arise in acute nephritis, which, after passing through the chronic stage, finally ends in cirrhosis; but in the great bulk of all cases the disease is an essentially chronic process from the very beginning. Gout and lead-poisoning are, beyond doubt, the cause of the affection in a considerable number of the cases; syphilis, gluttony, alcoholism, heredity, life at high intellectual pressure, and laborious physical strain may be also accepted as causal agents. Its relation to arterio-sclerosis will be discussed later on in this connection; it is enough to state here that the above-mentioned causes of general arterio-sclerosis may be accepted as the main etiological factors in this disease also. The disease is rare before middle life, and males are much more frequently affected than females. When

met with in young subjects it is regarded as a sequel to the acute form of Bright's disease.

Morbid Anatomy and Pathology.—The kidney may not reach half of the average normal weight; its capsule is thickened and adherent, and can only be removed by tearing the cortex; the renal surface is nodular and dark red in colour; numerous small cysts are visible, and the entire organ is firm and leathery. Upon section the cortex is seen to be much wasted, being represented by a thin line between the pyramids and the capsule. Under the microscope the intertubular connective tissue is seen greatly hypertrophied, especially towards the cortex, and in long-standing cases the shrinkage in this new-growth has caused such pressure upon the secretory parts of the organ as to cause widespread destruction of the tubes and their epithelium. The tubes which have escaped obliteration have their epithelial contents in various stages of degeneration—granular, hyaline, and fatty. The Malpighian bodies are destroyed in large numbers, many of them undergoing cystic degeneration, owing to the obstruction of their vessels; those which remain have thickened walls and show changes in their capillary tufts. The intertubular capillaries are more or less impervious through thickening of their coats, so that the blood-pressure in the glomerulus is continually raised, allowing of the escape or filtration of a large amount of water.

The arteries and arterioles of the gland show the typical sclerotic change in all their coats, which are thickened, the muscular tissue in the media being increased in some cases, and in others replaced largely by fibrous growth. The numerous small cysts which are present are caused by dilatation of portions of tubes which have been ligatured by contracting strands of fibroid tissue. The thickening of the coats of the blood-vessels throughout the body, and the cardiac hypertrophy, are identical with those observed in arterio-sclerosis.

There are different theories for the interpretation of the above-described pathological changes. Some regard the starting-point as a degeneration commencing in the secretory epithelium and glomeruli, to which the new-growth is secondary. Another view is that it is an inflammation of very chronic nature which produces fibroid growth akin to that seen in hepatic cirrhosis, and to the inherent contractile nature of this growth all the other changes are secondary. A third contention raises the important relation of this affection to general arterio-capillary fibrosis. It is accepted by most authorities that the form of chronic Bright's disease under consideration is but a local manifestation of the universal vascular change. In the typical form of small red kidney of chronic Bright's disease there are always found evidences of arterio-sclerosis in the

vessels throughout the body, and associated hypertrophy of the heart, so that this general cardio-vascular fibrosis must be regarded as a portion of the morbid anatomy of the disease. Dickinson stoutly maintains, however, that the cardio-vascular change is secondary or subsequent to the renal affection, and that this latter is produced by causes which act especially, and in the first instance, upon the kidney itself, and not upon the general vascular system.

Symptoms.—These are often unnoticed till a routine examination, made when treating some transient ailment, reveals the renal lesion. It may be first discovered by the examiner for life insurance, or by the ophthalmic surgeon, or it may be first suspected in the presence of some fatal result of the disease, as in cerebral apoplexy, when the *post-mortem* demonstrates it. Speaking roughly, the symptoms may be grouped under two heads—those which are the result of the cardio-vascular changes, and those which depend upon the retention of poisonous excrementitious products in the blood.

The symptoms caused by the condition of the heart and vessels are those described upon page 66. There is hardness of the pulse, with thickening of the radial coats, and the other signs of raised blood-pressure. The apex-beat is lowered and displaced towards the nipple-line, with general increase of the area of cardiac dulness; palpation reveals the heaving impulse of hypertrophy, and there is a marked accentuation of the aortic and pulmonary second sounds, and sometimes reduplication of the first at the apex. At a late stage signs of dilatation, with a systolic murmur and the usual symptoms of failing compensation, may appear, when the whipcord pulse, which easily rolls under the finger and can only be compressed or obliterated with difficulty, gives way to the pulse of low pressure in a thickened artery.

The circulation in the kidney, and the local lesions there, give rise to marked and characteristic changes in the renal secretion. It soon becomes increased in amount, pale or colourless, of low s.g., and in advanced cases this may fall under 1005; the patient is compelled to micturate two or three times at night and constant thirst is complained of, so that he may seek advice for what he fears is diabetes. Upon standing, or after spinning the sample, a scanty sediment containing a few hyaline or dark granular casts may be found, but these are often absent. Blood-cells are generally absent; but it is usual to find these, and all forms of epithelial casts and *debris*, in cases which have been exposed to chills and wettings a short time before the examination. The amount of urine may be double the normal, but the most constant and important sign is the low s.g.; as a rule, this may be said to be always lower than the dilution will account for, and in this fact lies the key to prognosis and treatment. It means that the

excretion of solids is constantly diminished, and that there is proportional danger of the onset of serious uræmia; but, as in the case of alcohol, morphia, and other forms of chronic poisoning, it is sometimes rather astonishing to observe the tolerance of the nerve-centres to the toxic agent. At a very late stage the amount of urine falls below the normal and the s.g. rises, the output of solids being still below the normal. Albumin is always small in amount, and is often entirely absent; perhaps, in reality, *it is never absent at all times in a given case*, if the examinations be made with sufficient care and frequency. It is usual to find no trace of albumin by a careful application of the cold nitric-acid test; but the observer may very often distinguish the characteristic ring or haze if he chance to look again at the test-tube after the expiration of many minutes, or an hour. This precaution is essential in dealing with the urine in this disease, and it is to be feared that it is often overlooked.

Just as in the very late stages the amount of the urine may diminish and the density increase, so the quantity of albumin may markedly increase when, owing to various combinations of circumstances and symptoms, the kidneys may become congested, or a tubal attack of nephritis supervene.

Dropsy is the next important feature for consideration. It is, like the albuminuria, always slight or almost imperceptible, and said to be often entirely absent; but when repeatedly looked for, from day to day or week to week, it is certain to be found some time in the course of the disease. At a late stage, as in the case of the albumin, it may be general and abundant, especially when failing compensation from dilatation has set in, with congestion of the kidneys and other organs. Slight anasarca, which is often overlooked, may be demonstrated by firm pressure on the middle of the sternum, or over the centre of the forehead, or under the conjunctiva, when the usual tibial site shows no traces of it. Towards the close of the disease the symptoms of embarrassed function caused by serous accumulations, and œdema of the lungs and larynx, may complicate matters.

Hæmorrhages are not unusual. The most important of these, from a diagnostic point of view, are the flame-shaped bleedings seen in the retina, which usually take the form of a stellate figure round the macula lutea, or of hæmorrhagic blots over the fundus, mixed with white patches caused by the deposition of fat-cells; they are found, however, in all the forms of nephritis.

Cerebral hæmorrhage is a striking feature of the disease, and it is its most serious and important complication; a large number of the victims of the small red kidney terminate their career in this manner.

Hæmorrhages into the lung, and from the nose or into the conjunctiva, are not rare; and Dickinson has described a common form of submucous hæmorrhage affecting the intestine near to the cæcum and ending in a circumscribed ulceration which often leads to fatal perforation. Uterine bleeding is not rare, and purpuric hæmorrhages from the mouth have been often recorded.

Owing to the injury of the epithelial elements in the tubules, urea and other products are retained to a greater or less degree in the blood, notwithstanding that the skin and bowel assist by their vicarious action the partially disabled renal organ. Though it is still desirable to speak of the symptoms so produced as uræmia, it must be always with the understanding that other retrograde products and other factors are also probably at work, so that the sum total of the result which we call uræmia is not simply the phenomenon of poisoning by urea. This part of the subject is dealt with in the article on 'Uræmia.'

Headache is generally at some time a prominent symptom. It may be transient and slight in the earlier stages, or intense in those cases where coma and convulsions are threatening; its character, duration, and intensity may indicate the degree of poisoning; and hence its importance. Dimness of vision of a transient nature, and independent of retinal hæmorrhage, indicates the action of the poison upon the nerve of sight, and it may amount to sudden total blindness, the presage of coma or convulsions.

Hearing is diminished at times, and all sorts of subjective disturbances, as tinnitus, noises in the ears, and vertigo, may be complained of. Neuralgic pains in various sensitive regions are to be traced to the same cause; transient paralysis is not uncommon towards the close; muscular twitchings are often the heralds of a general convulsion. The stomach often makes an attempt to eliminate the poison, and uncontrollable nausea and vomiting independent of food are the result. These may alternate with intractable dyspepsia. The vomited matter and breath may be ammoniacal; the vomit is generally alkaline. Respiration may become suddenly laboured, and asthmatic paroxysms, coming on at night, may present a formidable, and even fatal, group of symptoms, ending in hiccough or Cheyne-Stokes respiration. In this so-called uræmic asthma, the heart's action may be so disturbed as to represent the clinical picture of the cardiac dyspnœa and heart-block seen at the close of valvular disease.

Inflammation of the serous membranes caused by the impure blood circulating through their vessels is common, and pleuritis, peritonitis, and pericarditis may supervene; the latter is sometimes present with slight symptoms, and it is almost always fatal. Bronchitis and pneumonia are not uncommon.

Brain-symptoms, as delirium, mania, delusions, melancholia, and other forms of mental disturbance, may be present before convulsions and coma appear to wind up the scene.

All the symptoms mentioned may occur in any form of nephritis, but upon the whole a slowly and gradually deepening coma is more frequently seen in this form than are convulsions which are common in tubal nephritis. Possibly the long duration of the disease may so accustom the nerve-tissue to accumulating dosages of the toxic agent, whatever it may be, that coma is more likely to result than muscular movements.

Skin-eruptions are not rare, and various forms of papular rashes, eczema, dermatitis, and erythema have been observed; these are generally attended with much itching. Their appearance very often marks the last stage of the disease, and they deepen the prognosis of impending death. They are the result of the ineffectual attempt upon the part of the skin to act vicariously in the elimination of the urea, and sometimes urea-salts have been found as a white crystalline powder encrusting the entire body-surface.

Diagnosis.—When the disease has lasted for any considerable time, its symptoms and signs are so distinctive that there is little risk of its being mistaken for any other affection. The high-tension pulse, with its artery exhibiting to the touch of the finger its stiff and thickened coats, the presence of cardiac hypertrophy, and the increased amount of urine of low s.g., with a shade of albumin and no dropsy, are characteristic. The difficulty lies in those cases where there is no albumin. The cardio-vascular changes with polyuria are evidence sufficient, but albuminuria will almost certainly be found if repeated examinations be made ; in these cases the microscopic characters of the urinary sediment are of great importance, and the sphygmograph should be pressed into the service.

Where tubal nephritis supervenes upon the disease under consideration, the urine may exhibit the characteristics met with in the secretion found in the large white kidney ; such cases are very rare, and when met with they should be easily differentiated by their history.

Valvular disease of the heart may be so closely simulated by one stage of the cirrhotic kidney that error in diagnosis is very likely to occur. This is the period when the hypertrophied heart is no longer able to carry on the circulation of the impure blood through the obstructed and thickened arteries; dilatation gradually or suddenly takes place and mitral regurgitation, with its systolic murmur, follows as a secondary result from the inability of the valve to close accurately the dilated opening. Here all the signs and symptoms of failing compensation, following an old rheumatic valvulitis, may be seen, and the

difficulty is not lessened by the presence of dropsy and by the fact of albumin being found in the urine, since both these signs are common to the two conditions. Should this change to failure in compensation occur whilst the case has been under observation for some time, there will be no difficulty in its interpretation; but where the case is first seen with symptoms of dilatation, the difficulty is great. The pulse reveals low tension in the artery owing to the fall of pressure from cardiac failure, but with the educated finger the whipcord-like thickening and stiffness of the vascular walls will demonstrate arterio-sclerosis, and a painstaking analysis of the history of the case will enable the physician to come to a conclusion, assisted by the microscopic characters of the urinary sediment. The value of a careful ophthalmoscopic examination is obvious in such a difficulty: hæmorrhages, albuminuric retinitis, and papillitis or inflammatory swelling of the disc may be demonstrated.

Prognosis.—The end is certain if the patient be not carried off by some other affection; but the disease is essentially chronic, and for many years—sometimes for ten years or more—he may with care manage to lead a fairly comfortable life. He is, however, always open to the danger of uræmia, cerebral hæmorrhage, or inflammation of a serous membrane. In the early stage the prognosis as regards longevity, though never favourable, may be hopeful in proportion to the patient's power of changing his habits and environment in those cases where severe and prolonged mental strain, gout, lead, or alcoholism are the obvious causes.

Life may be prolonged, even after symptoms of failing compensation have appeared in some cases, especially when the immediate cause of the cardiac failure is a transient one, as after some prolonged muscular exertion.

Treatment.—There are few incurable conditions which the physician can approach with the same calm confidence of being able to prolong life and retard the natural progress of a disease which, though ultimately moving to a fatal issue, may be modified, or for a time arrested, by judicious management.

The causes which have set the diseased process in motion must be sought out, and when possible removed; in the case of lead, gout, alcohol, over-eating, constant exposure to cold and wet, and life under high physical and mental pressure, much may be done in arresting the progress of the arterio-sclerosis by removing the cause. The chief aim should be to so regulate the patient's habits that the least possible labour be thrown upon his kidneys, and that other organs should be trained to perform as large a share as possible of the renal duties.

Diet should therefore be light, and the nitrogenous elements reduced as far as practicable. Fish and poultry should take

the place of red meat, and every form of farinaceous and vegetable food may be permitted ; should the patient become a vegetarian, and include milk in his diet, all difficulty will be solved. Eggs and alcohol should be forbidden ; there is no objection to a liberal supply of liquids.

Residence in a warm equable climate is most beneficial; and when this is not possible, the patient may be induced to pass the worst English months abroad, in some region like Algiers or Egypt. Extremes of heat and cold, damp and east winds, should be guarded against by suitable clothing ; chills, sudden and prolonged, may at any time induce tubal nephritis upon the cirrhotic form. Moderate exercises should be the rule, and walking is best of all. The bowels should be carefully attended to, and saline purgatives, once or twice a week, may be freely prescribed. A short residence at a mineral spa for a portion of each summer is of great benefit, especially to those who cannot change their English climate for a drier and warmer one.

As regards the use of drugs directed against the diseased process itself, regular courses of iodides, for two or three months at a time, are unquestionably of considerable value ; and in the intervals between the courses the chloride of gold and sodium double salt, in doses of $\frac{1}{10}$ grain, is beneficial.

Warning is necessary against the indiscriminate use of diuretics and cardiac tonics of the digitalis type ; any drug which tends to raise blood-pressure, by causing further peripheral resistance in the small vessels, can only act injuriously unless in failing compensation. When a cardiac tonic is required, the combination of strychnine with strophanthus is preferable to digitalis, unless in the presence of great anasarca. As regards the routine administration of vaso-dilators similar warning is necessary ; they should not be employed unless when the continual high tension is indicative of danger, and then their use should be carefully directed and watched. A very low tension is to be feared almost as much as the opposite condition, especially if it be unwisely induced by injudicious dosage. The ordinary plan of giving large quantities of nitrites, which cause a sudden fall of pressure in the vessels, rapidly followed by a corresponding sudden rise, is to be condemned, as their short-lived action passes away long before the next dose is due. The blood-pressure is thus kept in a state of oscillation, which, in the opinion of the writer, is favourable instead of antagonistic to the progress of the diseased action. If vaso-dilators are indicated, they should be administered upon the plan laid down upon page 52. Erythrol tetranitrate, recommended by Professor Bradbury for angina, has a much more prolonged action than the nitrites. The usual physiological action—throbbing of the carotids, flushing of the face, and headache—should never be produced, except in the

presence of some urgent group of cerebral or uræmic symptoms. In serious dilatation, nitroglycerin may be given in full doses, in combination with digitalis and strychnine. Other complications must be met as they arise ; dropsy and uræmia should be promptly treated upon the principles mentioned in the preceding pages.

Anæmia will call for iron ; but often harm is done by indiscriminate dosing with this drug, which is so constantly valuable in tubal nephritis.

AMYLOID KIDNEY

Though, strictly speaking, the amyloid, lardaceous, or waxy degeneration of the kidney should not be included under the heading of chronic Bright's disease, it is dealt with here for convenience, and because its supervention upon the other forms of chronic renal inflammation is not infrequent, as *post-mortem* records show.

Etiology.—The degeneration is caused by the infiltration of the coats of the vessels, and of the various organs, by a protein body known as lardacein. Its distribution in the body is always universal, never being confined to any one gland. Certain factors always are found preceding its advent ; these are either syphilis, tuberculosis, or prolonged suppuration, from whatever cause, especially if of tubercular origin. The disease may occur at any age, the period of its incidence in a given case depending upon the primary cause ; thus, when it follows tubercular lesions it usually occurs earlier in life than when it supervenes upon tertiary syphilis.

Morbid Anatomy.—The organ, to the naked eye, is smooth and enlarged ; when long affected, it may be double the normal dimensions ; it is whitish in colour, and waxy-looking ; at a later stage still, it may shrink and be lobulated.

Upon section the cortex is seen to be greatly increased in size, and the Malpighian capsules easily recognised by their glistening appearance ; it is yellowish-white in colour, and contrasts markedly with the normal or deeply injected pyramidal portion. The characteristic iodine reaction is visible to the naked eye, even in those early cases where the organ may not be appreciably increased in size. The test is applied by moistening the cut surface with weak tincture of iodine, when the affected portions show a deep brown staining, the unaffected parts almost escaping. If a solution of iodine in water be used, the tissue must be soaked in it, and the lardaceous infiltration, after a time, stains most markedly without affecting the normal parts, giving a still more characteristic effect, as the contrast is more strongly marked. If, after the application of tincture of iodine, sulphuric acid be added, the stained parts are coloured instantly, almost black. Lugol's solution (5 per cent. iodine, dissolved in 10 per cent. iodide of

potassium solution) is the best iodine stain. Methyl-violet, when applied to a fresh section, stains the lardaceous material red, the tissues being coloured blue. Under the microscope the infiltration is seen to be almost entirely confined to the vessels ; beginning in the Malpighian tufts and their afferent vessels, it extends to the straight arterioles in the cones, ultimately involving all the small arterial walls, the veins escaping. The infiltration leads to thickening of the Malpighian capsule and increase of the intertubular tissue, which, in its turn, involves the tubes, so that the pathological changes observable in interstitial and tubal nephritis are added on to the amyloid condition ; but the tubes themselves and their epithelial contents usually escape the infiltration: these show the usual granular and fatty degeneration of their epithelium. Sometimes the membrana propria, however, exhibits the characteristic colour-reaction, and the casts found in the interior of the tubes often respond to the iodine test.

Symptoms.—The onset is always insidious, the symptoms being at first obscure or absent till an examination of the urine reveals the presence of the disease, which may be found to affect the liver and spleen also. The evidences of the primary disease—tertiary syphilis, phthisis, caries of bone, empyema, or other source of prolonged suppuration—may cause the renal symptoms to be overlooked ; but it must not be forgotten that the suppuration may have already entirely disappeared and left no mark behind it save the damage to the kidney or liver.

The patient will probably show some signs of anæmia or present a cachectic appearance ; he complains of slight thirst and polyuria, and he may have discovered some slight puffiness about his ankles. Diarrhœa may have led him to seek advice ; this has its origin in the lardaceous infiltration that sometimes invades early the intestinal mucous membrane. When dropsy is present, it is generally the result of anæmia, or, at a late stage, of tubal nephritis ; often it is never seen in the early stages. The characters of the urine are of great importance. It is markedly increased in amount in most cases, and may measure as much as one gallon in the twenty-four hours ; it very commonly approaches to half this amount—*i.e.* 80 oz. Its s.g. is low—generally somewhere about 1010. Though all the solids are slightly reduced in typical cases of the established condition which are still free from tubal or interstitial change, it may be roughly stated that the s.g. is almost in proportion to the amount of dilution ; at all events, this rule is nearer to the truth than is the case in the other renal diseases, and it gives the keynote to the symptomatology of the affection, since uræmia is consequently much less likely to occur. Towards the end of the disease, when other changes in the kidney are superadded, of course the secretory functions may

become seriously involved, and the urine may fall far below the normal ; its s.g. may then rise, and it may be loaded with albumin.

Albumin is always present, usually somewhat smaller in amount than is found in the large white, and always greater than is to be seen in the small red, kidney. Serum-globulin accounts for the great bulk of the albumin ; but as it responds to the ordinary tests for serum-albumin, which is also always present, its differentiation is of little importance, though Senator has shown that the globulin is constantly present in larger proportions than is found in other renal disease. There is very little urinary sediment. Upon spinning, a few casts may be obtained: these are of various sizes, the smaller ones predominating, and generally they present a transparent fibrinous or hyaline appearance; but every form of granular or fatty cylinder may be observed in very small numbers, if the examinations are frequent and exhaustive. Blood is, however, very rare, and there is little epithelial *débris*. The iodine reaction, as a rule, is not obtainable in testing the urinary sediment ; but occasionally a cast may respond to it, and sometimes the epithelial *débris* gives the characteristic reaction. Rare cases have been reported in which albuminuria was absent ; but had these patients survived long enough, it is highly probable that albumin would have appeared.

There are generally no cardiac signs, and the hypertrophy and increased blood-pressure and thickened vessels met with in chronic Bright's disease are rare ; but complications, as pleuritis, endocarditis, pericarditis, and peritonitis, are not uncommon. The most serious result of the lardaceous infiltration is the ulceration of the intestine which may supervene, causing death from exhausting diarrhœa. Retinal changes as in other forms of renal disease, are very seldom, if ever, met with.

Diagnosis.—The only difficulty will be in the exclusion of the other forms of chronic renal disease, and, as a rule, this can be overcome by an examination of the urine, supplemented by the history of the cause of the disease—*i.e.* syphilis, tubercle, or suppuration. The presence of a very considerable amount of albumin, with increase in the bulk of the urine, in the absence of thickening of the radial pulse and cardiac hypertrophy, distinguishes the disease from the cirrhotic kidney. The increased bulk of the urine, though the amount of albumin may be large, taken together with the absence of general dropsy, helps to distinguish it from the large white kidney disease. The presence of a uniformly enlarged liver and spleen in an anæmic syphilitic subject, or in one who has suffered from any form of prolonged suppurative disease, even when the urine is not typical, gives a clinical picture not likely to be mistaken.

Prognosis.—Though unfavourable always, it is more hopeful than in the chronic forms of nephritis, especially when it is seen in the early stages, and especially when the cause is syphilis. When of long standing, and where dropsy has supervened, the end will not be many months off, though syphilitic cases may recover under treatment after dropsy has been extensive. The disease may, however, last for several years, and its duration is much influenced by treatment ; in many early cases a cure may be expected upon the removal of the suppurative focus of the original disease. Occasionally the disease runs a rapid course, and may prove fatal inside half a year. Severe diarrhœa, indicative of intestinal ulceration, deepens the prognosis, and is always fatal.

When the urine has become scanty and highly albuminous, and the advent of considerable dropsy indicates that tubal or interstitial nephritis has supervened, the case is hopeless, if depending upon suppuration, as operative interference at this stage is not justifiable.

Treatment.—The first indication is the speedy removal of the cause of suppuration, and though the subject of an amyloid kidney generally bears operative interference much better than might be expected, still it is well to submit him to as little risk as possible, and often it may be wiser to sacrifice the part by an amputation than to perform excision of a large joint. Dickinson recommends that liquor potassæ be given before food, with the view of making up the loss of potash caused by the suppurative process in those cases beyond operative relief. Cod-liver oil and iodide of iron are always of value, and change to an equable climate is beneficial.

In syphilitic cases the hope lies in the prompt and persevering administration of iodide of potassium in large doses. Thirty grains may be given three times a day for three or four months at a time ; and during the month's interval, before renewing the course again, teaspoonful doses of the syrup of iodide of iron may be administered three or four times a day. Under this treatment the disease may entirely disappear, even when there is already marked hepatic and splenic enlargement. Complications, as they arise, should be treated upon the general principles laid down for the management of chronic Bright's disease.

BROMIDROSIS, or OSMIDROSIS

This is the name given to a fetid condition of the perspiration, generally accompanied by an excess of the secretion (hyperidrosis).

Etiology.—The offensive odour may depend upon a change before or after the perspiration has been secreted ; it is the

result of a decomposition of the sweat by the influence of *Bacterium fœtidum*, which is a common micro-organism found on the surface of most soils, and often upon the skin of healthy feet.

It occurs in young subjects, and is common in domestic servants. Nearly always the feet are the parts affected ; the axillæ and perinæum may also, to a much lesser degree, emit the foul-smelling odour.

Symptoms.—The insufferable stench renders the victim offensive to those about him, and often his boots and stockings retain the odour for days. There is little to be seen save a sodden or macerated condition of the skin, caused by excessive perspiration, and occasionally some blisters or bullæ are present. Often the patient himself is unaware of the odour, but may seek advice for the tenderness or even lameness which results from the inflamed or abraded skin between the toes and around the margins of the soles.

Treatment.—Absolute cleanliness is the first essential, and many cases can be robbed of their offensiveness by daily washing and changing the stockings twice in the working hours. After thorough cleansing in soap and water, the feet should be soaked in saturated solution of boric acid and permitted to dry by exposure to gentle heat. The stockings, having been previously soaked in the solution, should be thoroughly dried and dusted with the finely powdered acid which should also be packed between the toes. If care be taken to ensure the above routine twice daily, the odour in a few days entirely disappears. Cork in-soles, previously saturated with the solution, and dried, should also be worn, and shoes are to be preferred to tightly laced boots ; or where boots must be worn, they should have tops of light and porous cloth instead of leather.

Salicylic acid may be added to the boric powder, or a weak (3 per cent.) salicylic-acid ointment, or a strong boric ointment may be used. The application of a 5 per cent. chromic-acid solution is very efficacious, but it is dangerous, especially if there be blisters or ulcers present.

Neebe cures the condition by applying strong hydrochloric acid to the soles for ten minutes every three or four days.

Salicylic acid and sulphur internally assist the local agents. (For various other methods, see the author's 'Dictionary of Treatment,' 4th ed. pp. 92, 93, 699, 700.)

BRONCHIAL-GLAND DISEASE.

(See under MEDIASTINUM.)

BRONCHIECTASIS

This term is applied to dilatation of the bronchi.

Etiology.—Bronchiectasis has given rise to endless and conflicting hypotheses regarding its etiology. Into the relative merits of these various theories it is unnecessary to enter; suffice it to say that from some cause, either inflammatory, degenerative, mechanical, hereditary, or from loss of innervation, the wall of a bronchus becomes weakened, and the internal forces brought into play in the acts of breathing and coughing, or in the slow contraction from without of newly formed fibrous tissue formed in the lung, produce dilatation of the weakened tube. The continuous pressure exerted by a retained secretion, as when the main bronchus is involved by the proximity of an aneurismal or other tumour, may induce the condition in an otherwise healthy tube. The affection is found associated with, or as a sequela of, various diseased conditions—*i.e.* bronchitis, influenza, emphysema, pleuritis, pneumonia, pertussis, asthma, fibroid phthisis, the presence of foreign bodies, and more rarely tubercular disease and aneurismal pressure. Boggs lays stress upon the serious *rôle* played by the influenza bacillus in the production of the dilatation. There is evidence to show that in rare cases the condition may be congenital. Clinically, bronchiectasis is not to be confused with bronchiolectasis, an *acute* form of dilatation of the bronchioles or *small* tubes occurring throughout both lungs in children the subjects of acute bronchitis associated with widespread secondary pulmonary collapse.

Morbid Anatomy.—The dilatations may be either *cylindrical*, *fusiform*, or *saccular*. In about 50 per cent. of the cases of bronchiectasis the disease is confined to one lung, in which all forms of the dilatation may be present, and all the lobes may be the seat of the affection. The most typical and extensive dilatations are found in the bases of cirrhotic lungs, and often in association with chronic pleuritis. The saccular dilatations vary in size from the bulk of a grape to that of an orange, and more.

The mucous membrane lining the cavity may present the ordinary velvety characters of an inflamed bronchial surface; but at a later stage ulceration may be present, and gangrene often supervenes. In some cases the epithelial elements of the normal membrane are replaced by a smooth shining layer of flattened cells, as in ordinary cyst-formations. The changes in the original bronchial coats also exhibit variations in different cases; there may be simple atrophy or thinning of the walls, as in the dilatations met with in connection with emphysema, or the coats may be thickened from inflammatory action, which may extend into the surrounding pulmonary

interstitial tissue. The secretion found in the cavities varies from the clear mucus or muco pus of bronchitis to the most fetid purulent secretion laden with all sorts of putrefactive micro-organisms. Old dilatations are sometimes found with inspissated contents, in which lime-salts have been deposited.

Symptoms and Signs.—The disease is a slow and chronic process in its typical form, where its signs and symptoms gradually overshadow those produced by the affection which has led to the dilatation. All evidences of the original affection may have long since passed away when the bronchiectasis first comes under notice. A certain amount of dyspnœa is commonly present, especially on exertion ; but the chief symptoms are the cough and expectoration. Cough is always present, and generally characteristic in its peculiar intermittence and paroxysmal features. As a rule, whilst the patient is still there are no pulmonary symptoms till the cavity becomes filled with its purulent secretion, when a severe paroxysm of coughing is induced, and continues till the dilated bronchial cavity is pumped out. The intervals between the attacks of coughing vary very much in different patients ; as a general rule, the most severe paroxysm is that experienced in emptying the cavity in the morning after the night's sleep, and during the daytime the intervals are shortened. Change of posture, as stooping, or leaning over to one side, or any active exertion, may bring on an attack at any time by displacing the level of the liquid. The irritating nature of the secretion as it passes up the normal tubes causes such irritation as to prevent the patient checking his cough if he attempts to do so, and often distressing coughing may continue, with dyspnœa and cyanosis, after the secretion has ceased to come up, because of the basilar site of the cavity, which may refuse to be emptied till the patient bends his body forwards, or partially or completely inverts himself, when the residual pus may pour out without effort or noise.

The characters of the secretion are no less distinctive, though they may present considerable variation. The amount will depend upon the size of the cavity : a tumblerful may be brought up at once, but more commonly only one or two wineglassfuls are expectorated ; the daily quantity may exceed a pint, or approach to a quart, or even more.

In the early stages, and especially in small cavities situated in the upper or middle portions of the lung, where the contents are not permitted to lie for a long period, the secretion may be mucoid or muco-purulent, and free from putrefactive change. In most chronic basilar cases it is highly offensive and foul-smelling, and when permitted to stand in a glass vessel it separates into layers, the lower consisting of greenish-grey or brownish pus, and the uppermost of frothy opaque mucus. Under the microscope, pus-cells, bronchial epithelium,

and fatty-acid crystals are seen in abundance, and the liquid generally swarms with putrefactive bacteria. Charcot-Leyden crystals, sometimes blood-discs, threads of leptothrix, and even sarcinæ, may be found. Tubercle bacilli and elastic fibres are absent, unless there be active ulceration, when the latter may appear. The other physical signs will be described under Diagnosis.

Hæmoptysis is not uncommon, and it may be extensive at times. The breath is generally very fetid. The fingers are always clubbed, and the nails are incurved ; but the patient may show no signs of wasting or fever, though he is cyanotic and very short of breath. At a very late stage, fever and septic symptoms supervene from the absorption of the foul products of bacterial decomposition, diarrhœa, and sweating, with rigors and great cardiac depression, are common towards the close, which, however, may be long delayed.

Diagnosis.—The physical signs on inspection are generally sufficient to differentiate the disease from *phthisis*. The apex of the lung is generally free, whilst the favourite site of bronchiectasis is the base or middle of the organ ; in phthisis, where the lung is involved in its lower parts it is by extension downwards from the apex ; in the cirrhotic condition, associated with dilated bronchi, the change extends from the base upwards.

In the marked unilateral basilar cases associated with much fibroid change—the so-called cases of cirrhosis of the lung—the side is retracted ; the respiratory movements are diminished, and the heart is displaced in a horizontal direction towards the affected lung, and not obliquely upwards as in phthisis.

Percussion varies in its results according to the full or empty condition of the cavity, and this change is very distinctive. A region may be demonstrated to be dull which will give a clear tympanitic or amphoric note after a paroxysm of coughing which empties the dilated bronchus, and often a cracked-pot sound may be elicited.

Auscultation reveals similar peculiarities ; the breath-sounds may be entirely absent, but after coughing, cavernous or bronchial breathing, bronchophony and pectoriloquy, with amphoric râles, may be heard. The presence of emphysema may render the diagnosis difficult, especially in those cases where a considerable portion of one or both lungs may be involved with numerous sacculations, when the sounds will be those heard in bronchitis. Ewart lays stress upon the presence of '*croaking*,' a long-drawn semi-musical sound which is heard during the inspiration following a cough, and which is probably caused by the sucking backwards into the cavity of a plug of mucus partly expelled by the cough. It is the same as the *post-tussive suction* heard in phthisical cavities.

Empyema, which has ulcerated its way into a bronchus and been discharged by coughing, may resemble a dilated basilar bronchus, but the history of an acute illness with little or no expectoration, suddenly complicated by the presence of a large flow of pure pus without mucus, is so characteristic as to require little debate.

Gangrene of the lung is not likely to be mistaken for bronchiectasis, if a careful scrutiny of the history be made, the symptoms generally coming on suddenly, the sputum being characteristic in its odour and containing often much necrosing lung-tissue as described under 'Lung Gangrene.'

Congenital heart-disease, with its cyanosis, clubbed fingers, and dyspnœa, makes a picture closely resembling bronchial dilatation, but the history settles the diagnosis. The writer had once a case of bronchiectasis in a boy presented to him fraudulently for life-insurance, with a suppressed history; there were no physical signs to be discovered in the chest and no cough was present, but slight cyanosis and some clubbing of the fingers strongly suggested congenital heart-disease. The foul odour detected from the breath aroused suspicion of bronchiectasis, and by making the candidate stoop down and touch the ground with his fingers, half a tumblerful of foul pus was expelled by violent coughing, after which a clear area was demonstrable at the right base close to the normal hepatic dulness.

The chronic history and slow progress of bronchiectasis, the fair condition of the patient's nutrition, the absence of tubercle bacilli, of fever and hectic symptoms, as well as the physical signs already enumerated, easily differentiate the disease from tubercular phthisis even in its most chronic forms.

Prognosis.—This is more or less serious when the disease is established, and it becomes grave when the cavities are multiple; and even when it is unilateral and associated with cirrhosis of the lung, the most simple chronic cases may, owing to the supervention of heart, liver, or kidney disease, pneumonia, pyæmia, or sepsis, become serious at any time. The chances of cure without radical treatment are nil, except in the acute cases of childhood. The disease may continue stationary for very many years, and by careful attention to the disinfection of the cavity it may sometimes be so modified as to have practically no effect upon life. Profuse hæmoptysis may be fatal.

Treatment.—Cod-liver oil and a plain highly nutritious diet are indicated. The condition of the cavity demands attention, and every effort should be made to render this as near to aseptic as possible; volatile antiseptics are the only drugs of value. Capsules containing 3 mins. creosote, eucalyptol, guaiacol, or myrtol may be used, or terebene in 15-min. doses.

Garlic, as recommended by Poore, in cachets each containing half a teaspoonful of the chopped bulb, is the best of internal remedies.

Chaplin's method of treating the disease by inhalations of coal-tar creosote, heated in a suitable chamber (see 'Dictionary of Treatment,' 4th ed. p. 94) has given most excellent results, many cases being permanently cured. Intratracheal injections of menthol 5 grs., guaiacol 1–2 mins., in a teaspoonful of olive oil twice daily, through the glottis, are advocated by Ewart and others who have published favourable results; but Chaplin's method is better and safer, and has rendered surgical operation unnecessary and unjustifiable in most cases.

The plan of educating the patient to assist himself in emptying his cavities by assuming a suitable stooping posture is of the greatest importance as an adjuvant to all treatments. The relief of the catarrh, emphysema, and other concomitants is to be carried out by suitable climatic and other agents.

BRONCHITIS, ACUTE

Acute inflammation of the bronchial mucous membrane varies in importance and gravity in proportion to the size of the tubes involved in the attack. It is the site, and not any marked variation in the inflammatory process itself, which causes the different types of the affection. The term 'Acute Bronchitis' is generally applied to those cases where the large and middle-sized bronchi are selected by the inflammatory process. When the smaller tubes are involved in the adult, the affection is often called 'Capillary Bronchitis,' but this term should be reserved as a synonym for the condition met with in infants where the inflammation extends into the intralobular bronchioles and alveoli, when the affection is described as 'Broncho-pneumonia.'

Etiology.—Acute bronchitis is attributable to various causes, the most common being exposure to cold, especially when the air is laden with finely divided moisture, as in mists and fogs; when other impurities, as irritating vapours, smoke, dust, &c., are added the risk of attack is greater. Exposure of the surface of the body to any chilling influence is also a potent factor; hence the frequency of attacks in winter and early spring when east winds are prevalent. Certain diseases, as measles, pertussis, influenza, and Bright's disease, render the patient peculiarly susceptible. Often it is a simple extension downwards of a nasal or laryngeal catarrh, or the local manifestation of the ordinary epidemic microbic cold or catarrh. Powerful predisposing causes are emphysema, valvular lesions, residence in overheated, ill-ventilated rooms, or working in

factories where the warm moist air is laden with particles of fine dust. The disease is most common in the very young and in the aged.

Morbid Anatomy.—The changes are those characteristic of catarrhal inflammation, and vary with the stages of the process. The inflammatory action is ushered in with acute congestion of the mucous surface, which is swollen and dry; soon the basement-membrane becomes swollen, and the different layers of the bronchial wall become thickened and infiltrated with leucocytes. The epithelium is rapidly shed; the ciliated cells and the desquamated cells from the bronchial mucous glands and immaturely formed epithelium, and leucocytes or pus-cells, constitute the characteristic sputum, which is expectorated during life and found in the tubes after death. The redness and swelling of the tubes are best marked about their bifurcations and are rarely seen in the very small tubes. More or less pulmonary congestion, œdema, collapse of areas of lobules, and emphysematous patches are often present; the right cavities of the heart are distended with dark or black blood.

Symptoms.—The first symptoms of the attack vary much in different individuals. Generally there are mild constitutional disturbances, as slight feverishness, malaise, dry skin, loss of appetite, and backache; these are soon followed by local or chest symptoms. Tightness or a sense of oppression in breathing is complained of, and cough sets in; it is hard, dry, and painful at first, and associated with a distressing sensation of 'rawness' or pain behind the sternum, which may be so severe as to cause the patient to struggle against the inclination to cough, which generally aggravates the paroxysm and increases his distress. At this stage, which corresponds to the dry state of the membrane and skin, there is no secretion or sputum; gradually, after some hours, a little clear mucus is brought up, and as expectoration becomes freer the sputa become opaque, white, and frothy from admixture of air in the tubes. After a few days, owing to the presence of leucocytes or pus-cells, the secretion becomes muco-purulent, and finally purulent, greenish yellow, free from air-bubbles, and assumes the nummular form. At any stage, but especially in the early period, some fine streaks of blood may appear in the sputum, but they have no significance.

The physical signs show in mild cases upon inspection a slightly increased respiration-rate; in severer types the chest is uniformly over-distended, and the accessory respiratory muscles are in evident action; the respiratory movements are quickened, but less extensive in range, and dyspnœa may be present. The heart is slightly depressed, and generally there is epigastric pulsation. Upon palpation, bronchial fremitus is generally felt, and there is no increase in vocal

fremitus. Percussion generally gives a clearer note unless at the bases when there is abundant secretion. Auscultation reveals the absence of the vesicular murmur, which is obscured or replaced by rhonchi and râles. The rhonchi, which are dry musical sounds, are either sonorous or sibilant; the former are low-pitched sounds produced in the larger tubes; the higher-pitched, piping, or whistling rhonchi are produced in the smaller bronchi. Both may be heard in inspiration or expiration, and they constantly change their position and characters upon coughing or modifying the respiratory act. The râles are moist sounds with a bubbling character, and are, according to their coarseness or fineness, produced by the air bubbling through the secretion in the larger or smaller tubes. They lack the metallic, crepitating, or clicking character of the crackles heard in capillary bronchitis. The finer râles are generally limited to inspiration, but the coarser ones are heard during both acts. When the trachea and largest bronchial tubes are alone involved, there may be no adventitious sounds heard in the chest.

When the disease has extended into the finer tubes in the acute bronchitis of adults, the type is known as *Acute Suffocative* or *Capillary Bronchitis of Adults*, though it may occur in the aged. The invasion of the smaller tubes may be marked by a series of chills, with pyrexia. Sometimes, however, the larger and smaller tubes are affected simultaneously. All the previously mentioned symptoms deepen, the breathing becomes laboured, and the dyspnœa is expiratory, the chest being obviously overfilled with air; asphyxia, with lividity, results from the diminished aëration of the blood owing to the blocking of the bronchioles.

The physical signs on inspection and palpation are those of emphysema, with lowering of the diaphragm and descent of the heart and liver, with epigastric pulsation; the entire thorax is hyper-resonant, and fine bubbling râles are heard all over the chest, but best marked at both bases; there is absence of the loud sonorous rhonchi produced in the larger tubes. Cough is difficult; the expectoration is often abundant, consisting of white frothy mucus filled with fine air-bubbles; but as progressive difficulty in breathing, cardiac dilatation, anasarca, cyanosis, and mild delirium appear, the sputum diminishes. At first the patient sits up in bed bringing every accessory respiratory muscle into play, but gradually, as the asphyxia increases, the lower intercostal spaces fall in during inspiration, coughing becomes impossible, and expectoration ceases; he lies upon his side with the head depressed, and gradually falls into coma. There is rarely much fever and the temperature in the later stages drops below normal; the respirations may reach 60, and the pulse 140; there are generally profuse cold perspirations and the breath becomes cool.

Death may occur after forty-eight hours ; more frequently life is prolonged for several days.

The Capillary Bronchitis of Infants is described under 'Broncho-pneumonia.'

Diagnosis.—As a rule this is easy, and not likely to be mistaken for *pneumonia* owing to the lower temperature, the presence of the loud sonorous and sibilant râles and the bubbling rhonchi, and the absence of dulness and bronchial breathing indicative of pulmonary consolidation. The *capillary* or *suffocative* form is differentiated from the simple bronchitis involving the larger and middle-sized tubes by the grave constitutional disturbance, the dyspnœa, lividity, and cardiac frequency, the frothy or foamy character of the sputum, the presence of fine liquid crackling or bubbling râles at the bases, and the absence of rhonchi. This grave condition closely resembles the *capillary bronchitis of infancy* and *old age*, which is in reality a diffuse broncho-pneumonia ; but the age of the patient and the evidence of pulmonary collapse, with diffuse broncho-pneumonia, serve to distinguish the latter.

Secondary bronchitis coming on in the exanthemata, in Bright's disease, valvular affections, emphysema, gout, bronchiectasis, and many other diseased conditions, is often very insidious. It is easily, as a rule, differentiated by the history of the case and the other evidences of the underlying cause.

Prognosis.—In simple acute bronchitis affecting the larger tubes, recovery takes place generally inside a fortnight in healthy adults. When the bronchitis occurs in a patient already suffering from Bright's disease, diabetes, or cardiac disease, the prognosis is always serious, whether the bronchial inflammation be of the secondary type or the direct result of exposure. The outlook must be also guarded in the case of very young children and aged folk.

The acute suffocative form is often fatal, and almost always so in the presence of cardiac affections and in emphysematous and albuminuric patients, when death generally follows inside a week. In weak children the presence of measles and pertussis adds greatly to the gravity of the disease, which is liable at any time to run into broncho-pneumonia.

Treatment.—Mild attacks involving the large bronchi and trachea require little treatment ; as a rule, a few days' confinement to one room, a smart saline purge, following a hot bath, and, if necessary for the relief of pain, a large poultice to the chest, are all that are required. When the medium-sized tubes are involved the same measures are indicated. Much has been written about the plan of causing the abortion of the attack by large doses of quinine, iron, or other drugs, and by the use of snuffs, sprays, insufflations, and inhalations, or by the Turkish bath. As a rule, this plan is useless and ends generally in loss of time ; it is best to put the patient to bed

after a hot bath, in a room heated to 60°–65° F., the atmosphere of which is kept moist by a jet of steam from the ordinary bronchitis-kettle placed upon a fire in the open grate. A large linseed poultice, to which a little mustard has been added, gives considerable relief. Hot diluent drinks, as barley-water and linseed infusion, have a soothing effect where the pharynx is irritable, and all diet should be given in liquid form. The object should be to establish mild diaphoresis, for there is good reason to believe that as soon as the cutaneous surface becomes covered by perspiration the bronchial membrane rapidly passes through its dry stage, thus relieving irritative, painful, and useless coughing. Much may be done by judicious medication at this early period. Drachm doses of bicarbonate of potassium, with $\frac{1}{16}$ gr. tartar emetic, may be given in effervescence with fresh lemon-juice, and if painful cough is frequent $\frac{1}{20}$ gr. morphia may be added; morphia alone often aggravates, and should not be given to children. With a high, bounding pulse and much feverishness, 5-min. doses of tincture of aconite may be given, especially if there be nausea or vomiting. When the bronchial secretion is freely established it should be encouraged rather than checked, and small doses of ipecac. with acetate-of-ammonia solution may be administered. The common mistake of drugging with opiates at this stage is to be condemned. Should there be any difficulty in getting up the abundant secretion, either from want of expulsive power or from a viscid condition of the mucus, an emetic should occasionally be given; the best is ipecac., especially in the case of children, and in the case of the aged carbonate of ammonia or sal-volatile may be selected. One medium dose of opium, if given under these circumstances, may be fatal when the tubes are full of adhesive secretion.

At a later stage, when it is desirable to assist nature and check the purulent secretion by altering the action in the inflamed mucous membrane, there is no drug so universally beneficial as the iodide of sodium given with an alkali in moderate doses.

In the suffocative bronchitis involving the finer tubes, if life is to be saved, more active measures must be speedily employed. There is here no time for the ordinary diaphoretics to act: antimony is too depressing, and morphia is absolutely contra-indicated. The patient should be surrounded by an atmosphere saturated with moisture by means of the free use of steam. The main indication is the serious state of his blood-aëration owing to the blocking of the fine bronchioles and air-cells; and to meet this, oxygen should be administered early and continuously in conjunction with the inhalation of warm moisture-laden air. Expectorants at the early stages may do harm, especially where the secretion is abundant

and liquid and iodides are to be avoided. . The best method of relieving the block is to give a full emetic of carbonate of ammonia and, whilst waiting for its action, to administer a full hypodermic dose of strychnine ($\frac{1}{15}$ gr.). All through the attack the condition of the heart is of primary importance, and when increased lividity and failing rapid pulse show that its right chambers are overtaxed and are dilating, a large vein should be opened without delay or hesitation, and a distinct impression rapidly made upon the circulation. Cardiac tonics, the best of which is a combination of digitalis and strychnine, should be given every three hours, alternating with drachm doses of sal-volatile administered with an equal amount of sweet spirit of nitre to prevent increase of peripheral resistance through the digitalis action.

Alcoholic stimulation is generally necessary, but it must be used with caution so that the respiratory centre be not depressed ; occasionally one sees the deadly action of the drug when it has been pressed so far as to extinguish coughing-power, under which circumstances it may be as fatal as morphia.

Expectorants are indicated where the secretion is scanty or adhesive, and these can be judiciously combined with a drug whose action will tend to allay bronchial spasm. Belladonna is frequently prescribed with this intention, but it generally fails to give any relief, and it may dry up the secretion and do much harm. Tincture of lobelia may be combined with ipecac., and if pushed it will be found reliable in lessening bronchial spasm and aiding the expulsion of the bronchial mucus. Iodide of sodium may be advantageously pushed in the face of a scanty, adhesive secretion.

Ewart urges the importance of mechanically aiding the expiration in the emphysematous patient, where the danger of suffocation is always very great. This can be safely done by pressing with the spread-out hands upon the lateral aspects of both bases below the axillary regions during spontaneous expiration.

BRONCHITIS, CHRONIC

This may be the result of one or more acute attacks, but its most common form is seen in the catarrhal cough which affects elderly people each recurring winter in cold damp climates, and which passes away with the approach of dry summer weather. Between this mild type of the disease, often known as *winter cough*, and the severe forms of bronchorrhœa there are several clinical varieties ; but these present mainly differences in degree, the pathological factor being a chronic inflammatory change in the bronchial mucous membrane leading to thickening of the bronchial coats and, at a later

stage, to atrophy of their tissues and shedding of the epithelial cells ; there is always pulmonary emphysema present and more or less dilatation of the tubes in all long-standing cases.

The symptoms are those seen in the acute variety after the feverish stage of the attack has passed away. There is cough, with some dyspnœa and expectoration. This latter varies in character and amount, and constitutes the chief distinction between the clinical varieties of the disease. The physical signs are those of simple mild catarrh of the larger and medium-sized tubes—*i.e.* some degree of hyper-resonance, sibilant and sonorous rhonchi, and râles of different degrees of fineness, heard generally during the expiratory act which is prolonged, plus the signs of more or less emphysema with hypertrophy or dilatation of the right side of the heart. This latter condition ultimately leads to permanent embarrassment of the breathing, congestion of organs, some dropsy, and serious constitutional weakness or debility.

In ordinary *winter cough* the sputum varies from week to week, and the cough is in proportion to its viscosity, being very severe and often paroxysmal in the mornings when it is adhesive ; when muco-purulent or purulent, and profuse, it is got up with less labour. *Dry catarrh* is the name given to that form of chronic bronchitis characterised by difficult and continued paroxysms of coughing with little or no secretion ; it is always associated with the breathlessness and wheezing characteristic of emphysema. It is commonly seen in old people, and is almost universally present amongst the operatives in flax-mills. Any little secretion pumped up, generally consists of clear mucus in the form of pearl-like pellets, or sago-grains, which may be found to contain the crystals and spirals described under ' Asthma.' The gouty poison seems to induce this form of chronic bronchitis, which is always associated with more or less bronchial spasm.

Bronchorrhœa is the name given to chronic bronchitis characterised by the expectoration of large quantities of secretion, which may amount to 60 or 80 oz. daily. In one form, sometimes described as *serous* bronchorrhœa, the secretion is watery and transparent ; more frequently, however, it is *purulent* and semi-fluid, and liable to be mistaken through carelessness for bronchiectasis secretion. In some cases the secretion is ropy, and upon an attempt to transfer it from one vessel to another it pulls out in long gluey strings, which may measure several feet in length. The bronchorrhœic form of chronic catarrh is the most obstinate to treatment, and, though life may be maintained for many years, the inevitable emphysema and cardiac changes lead to very serious pulmonary embarrassment.

Putrid bronchitis.—A fetid or putrid condition of the sputum is constant in gangrene of the lung, bronchiectasis,

and some forms of phthisis and empyema; but it is generally held that the condition may supervene upon an ordinary purulent bronchorrhœa where none of these complications are present. Its occurrence is explained by the appearance in the putrid secretion of various putrefactive bacilli. The sputum separates into several layers on standing, the lowermost of which contain the so-called 'plugs of Dittrich,' which are dirty, greyish masses with foul odour, varying from $\frac{1}{20}$ to $\frac{3}{4}$ inch in size, and made up of pus-cells, epithelial *débris*, crystals, and numerous varieties of micro-organisms and oil-globules. There is doubt about which of the bacilli should be accredited with the production of the foul odour. When this putrid condition supervenes upon chronic bronchitis, there is a sudden deepening of all the symptoms; often pneumonic changes in the pulmonary tissue and typhoid symptoms of septic poisoning soon show themselves and end in a fatal issue.

Diagnosis.—The diagnosis of chronic bronchitis is as a rule settled by the history of the illness and the signs of a bilateral bronchial inflammation associated with emphysema and possessing no marked pyrexia; these sufficiently differentiate it from *phthisis*. Difficulty may be experienced in those cases of chronic bronchial catarrh limited to the very large tubes, and not an infrequent sequela of a long-past acute attack; in these there may be no physical signs heard in auscultation, but the nature of the clear sago-like expectoration and the distinct history of the annual winter cough clear up the difficulty.

Prognosis.—This will depend upon the ability of the patient to change his environment and winter in a warm equable climate; or, where a long journey or a foreign residence is impossible, much will depend upon his being able to protect himself from the variations of the cold moisture-laden atmosphere prevailing in the British Isles during the winter months. Life may be prolonged to old age in the case of the subject of bronchial catarrh who can protect himself by breathing a pure, indoor, moderately moist atmosphere during the periods of fogs, mists, or east winds; and in most instances where emphysema, bronchiectasis, and cardiac dilatation have not set in, a cure may be counted upon. Where such changes have already set in after years of recurring winter cough, in those whose means do not permit of climatic treatment or indoor life, the outlook is grave and the duration of life considerably shortened.

Treatment.—In the author's 'Dictionary of Treatment,' on pp. 95–107, the details of the treatment of bronchial inflammation are discussed; only a brief enumeration of the principles need be here mentioned.

Climate is of first importance. In Ireland, Rostrevor; in England, Torquay and Bournemouth, are favourite winter resorts. Abroad, Mentone and San Remo suit many cases; but where a *dry* warm climate agrees best, Egypt, Algiers, or Tangiers may be selected. Those patients with irritable bronchi and much coughing will find Madeira, with its soft sedative atmosphere, most advantageous.

Topical treatment by sprays, inhalers, and respirators gives very unsatisfactory results as a rule, but they are of undoubted value in the acute exacerbations following exposure, and in these counter-irritation may be advantageously employed.

Expectorants, unless when very judiciously employed, are productive of much harm, and unfortunately the popular idea of expectorant remedies consists of the notion that coughing should be checked or abolished by narcotics like morphia, chloral, &c. Narcotics should never be employed except when the coughing is out of all proportion unnecessary for the bringing up of scanty secretion; and when prescribed with the idea of lessening such needless expenditure of respiratory force, they should always be combined with a nauseating expectorant like ipecac. Iodides are the best routine agents for altering the diseased action and liquefying the sputum; they should be combined with alkalies and small doses of arsenic.

In the dry catarrhs, iodide of sodium in combination with full doses of ipecac., in courses alternating with slightly nauseating doses of apomorphine, gives best results.

In bronchorrhœa, the best line of treatment will be found in the administration of those volatile expectorants which are excreted by the bronchial surface. The best of these are tar, garlic, asafetida, copaiba, sulphur, terebene, and ammoniacum; they can be combined advantageously with ammonia and senega, which assist the mechanical expulsion. When the coughing power is failing, strychnine is indicated in full doses with the view of stimulating the respiratory centre and strengthening the cardiac fibre. Bronchial spasm must be met by lobelia and the drugs mentioned under 'Asthma.'

It is needless to say that the cause of the catarrh should be removed, if found out; thus, in the mechanical bronchitis produced in many industrial occupations, where the air is laden with particles of fine dust, change of occupation, if adopted in time, will often cure the disease. In the same way the treatment of gout, renal disease, and valvular lesions may entirely remove the secondary bronchial trouble. Cod-liver oil is beneficial in almost every case of bronchial catarrh of long standing, and iron is indicated where the profuse secretion leads to anæmia. The cardiac condition will at times demand the use of tonics of the digitalis group; these may be combined advantageously with ammonia and nux

vomica, and sometimes, though rarely, alcohol may be beneficially pressed into the service.

Diet, exercise, clothing, the state of the stomach, kidneys, and bowel, and such complications as insomnia, hæmoptysis, &c., are to be regulated by the application of general principles.

BRONCHITIS, PLASTIC

This affection, also known as Fibrinous or Croupous Bronchitis, is characterised by the expectoration of firm, arborescent, continuous moulds of considerable portions of the bronchial tract.

Pathology and Etiology.—Nothing is known of its pathology or causation, and a repetition of the speculations upon this subject is of no practical value. The casts are expectorated in rounded plugs or masses covered with mucus which unfold themselves when placed in water, or mere detached fragments may be expectorated. The cast at its thickest part may be as large in diameter as an ordinary writing-pencil; it is white, consists of stratified layers of fibrinous material, with a hollow centre, and shows sometimes a perfect mould of the smaller ramifications of the arborescent arrangement making up a portion of the bronchial tree. Under the microscope their structure is laminated, and is made up of epithelial, pus, and gland cells, hæmatoidin crystals, Curschmann's spirals, and various bacteria and oil-droplets embedded in a structureless, fibrinous, or mucin substance. Several protozoon bodies have been recognised, probably of accidental source; blood-corpuscles, when present, are generally found on the outer layer. The disease is very rare, it may appear at any age, and has been met with sometimes in several members of the same family.

Symptoms.—In its beginnings it is not to be differentiated from ordinary bronchial catarrh. The casts may be expectorated in fragments every day for weeks: more frequently they are retained for some days and then expectorated, so that the attacks of cast-expectoration come on with more or less regularity every five or six days, in the intervals ordinary bronchial mucus being brought up; but the greatest variations in this respect have been recorded, and the disease may last with irregular intervals of apparent convalescence for very many years.

The cough is generally violent when the casts are large, and much difficulty may be experienced for hours before the plugs can be dislodged; dyspnœa, cyanosis, and some hæmoptysis being common. Relief for a time is complete, depending upon the rate of the coagulation-process and the amount of bronchial tract involved. Examination of the

chest may reveal impairment of the respiratory movements confined to a particular region of the thorax, and this region may be the one always involved ; it may be marked by dulness and absence of vesicular murmur, showing temporary pulmonary collapse ; or loud râles or sibilant rhonchi may be heard. A peculiar flapping sound of the nature of a coarse dry râle has been described as pathognomonic of the loosening of the tail portion of the bronchial cast.

Sometimes the disease appears in an acute form during the course of a subacute bronchial attack secondary to some fever, and then the case assumes a grave type ; death may occur from asphyxia before the casts are expectorated. These are probably, however, instances of some other obscure pathological condition, possibly of diphtheroid nature. The casts have been described as found *in situ* in bronchial tubes, whose epithelial lining is intact, or has been already shed. The usual morbid characteristics of bronchial inflammation are found in such cases and emphysema is always present, and sometimes pneumonia and tubercle may co-exist.

Diagnosis.—The examination of the expectorated masses by unfolding them in water renders the diagnosis almost certain even to the naked eye ; further scrutiny by the microscope excludes blood-clots occasionally expectorated after hæmoptysis, diphtheritic membrane, pneumonic and asthmatic sputa.

Prognosis.—This is grave in the acute cases ushered in with fever, great dyspnœa, and signs of much pulmonary collapse, with very little sputum. In the chronic cases there is little injury to health and no danger to life, which may be prolonged into old age.

Treatment.—No specific treatment is known. The general indications are those of ordinary bronchitis, and much may be done by means of expectorants like iodides, tartar emetic, pilocarpine, creosote, or tar. Emetics of the ipecac type, combined with ammonium carbonate, may assist in the expulsion of the obstruction, especially after the sputum has been liquefied by pilocarpine or iodides.

Steam, or a warm moisture-laden atmosphere, cannot fail to assist nature ; and sprays of lime-water, which has been proved to be of value as a solvent of the casts, have proved of value ; with the same object in view, solutions of papain, pepsin, trypsin, and lactic acid have been recommended.

BRONCHO-PNEUMONIA

This disease consists of an acute interstitial inflammation of the terminal bronchioles and their adjoining air-cells, and is most frequently met with at the extremes of life. It is also known as Capillary Bronchitis, Catarrhal Pneumonia, Lobular

Pneumonia, and Disseminated Pneumonia, and less frequently by the objectionable and confusing title of *Pulmonary* Catarrh.

Etiology and Pathology.—Two forms of the disease are recognised—viz. the *primary* and *secondary*, and the causal factors at work differ in each case. In the *primary* form the etiological factors may be accepted as those present in ordinary acute lobar or croupous pneumonia, with the exception that the disease is practically confined to childhood. Thus, there is a sudden invasion following exposure to cold, and there are good grounds for believing that in the great majority of all cases the infective organism is the pneumococcus, though other organisms are also sometimes present.

In the *secondary* form, whilst the pneumococcus is sometimes present, there are almost invariably other microbes at work as well, so that the generally accepted view of the origin of this form of the disease is that of a multiple infection in which the pneumococcus need not necessarily figure. The following organisms have been found in the blood, lung-tissue, and organs, alone or with the pneumococcus—viz. *Streptococcus pyogenes*, *Staphylococcus aureus*, *Bacillus pneumoniæ*, *Bacillus diphtheriticus*, and sometimes the tubercle microbe and the organisms causing the primary affection. In this connection it may be mentioned that Dawson Williams has shown that the broncho-pneumonia of measles is itself infectious.

Secondary broncho-pneumonia, like the primary, is almost always a disease of infancy and early childhood, but it occurs also, though rarely, in old age in debilitated individuals.

The disease is secondary :

1. In children to the *acute bronchitis* which has attacked in the first instance the larger or medium-sized tubes, from which it extends into the bronchioles and lobular tissue ; or it may commence in the latter, when from any cause the lobules have collapsed.

2. The affection may be secondary to *whooping-cough*, *measles*, *influenza*, *diphtheria*, *rickets*, *scarlatina*, and *inflammatory diarrhœa*.

3. In patients at any later age who are suffering from typhoid fever, ulcerative endocarditis, variola, erysipelas, Bright's disease, the last stages of pulmonary phthisis, or other debilitated condition, though in all these affections the lobar type of pneumonia is far more frequently to be met with.

4. Broncho-pneumonia produces the characteristic lesions in the lung which have been designated aspiration-pneumonia, and which owe their origin to septic particles passing through the paralysed larynx after operations on or during the progress of malignant disease in the upper air-passages. This cause, like the last-mentioned, may operate at any period of life.

5. In aged subjects, debilitated from any cause whatever, the disease, though rarer than in children, is often seen as a

terminal event in ordinary bronchitis, Bright's disease, and in every bed-ridden condition in which hypostatic congestion is liable to occur from prolonged posture on the back in patients confined to bed from hip-joint traumata, &c.

Morbid Anatomy.—Since the starting-point in many instances is the acute bronchial inflammation affecting the larger or medium-sized tubes, the changes characteristic of acute bronchitis are commonly also present. The essential broncho-pneumonic lesion consists of changes observed in the walls of the terminal bronchioles, and in the air-cells or alveoli which branch off from them. To the naked eye, the normal infundibula upon the surface of an inflated lung, which represent the smallest ultimate lobules of the organs, are visible as small areas made up of a series of air-cells, or pulmonary vesicles, each air-cell measuring about $\frac{1}{100}$ inch. These areas are polygonal, and each encloses a group of six or eight air-cells. An inflamed lobule is converted into a firm, solid, airless mass which, when upon the surface of the lung, projects slightly above the level of the surrounding healthy tissue and preserves its polygonal outline and exhibits a light-red or greyish-red colour. Collapsed lobules, which have not become inflamed, appear as depressed areas of a dark-blue or purple colour, especially in the basal region scattered amongst the raised nodular patches. The inflamed lobules, at first isolated, tend to coalesce, so that large patches or tracts of solid pulmonary tissue are found scattered along the posterior margins and bases of the lung and disseminated throughout its structure. The patch made up of the coalescence of a number of inflamed lobules varies in size, and in some instances may apparently involve the greater part of an entire lobe; more frequently, however, the patches are irregularly distributed throughout both lungs, and are divided by considerable tracts of healthy pulmonary tissue.

Upon section the lung is seen to be of a dark mahogany colour, and blood oozes from the cut surface, which here and there exhibits the characteristic lighter-red or greyish isolated or coalesced inflamed lobules; these, as upon the free surface of the lung, are observed to project slightly above the level of the section.

Under the microscope the walls of the terminal bronchi are seen to be swollen, and the seat of interstitial inflammatory changes as evidenced by the extensive cell-infiltration present, and more or less peribronchial inflammatory change penetrates into the surrounding tissue, accounting for the hepatisation and splenisation observed by the eye in a section. Similar interstitial inflammatory changes in the swollen alveolar walls are invariably present, the air-cells being distended with epithelial elements and leucocytes, the alveolar wall itself showing extensive leucocytic infiltration and enlargement of its

capillaries. The lumen of the terminal bronchioles is filled with a mass of swollen epithelial elements and leucocytes, which to the naked eye resembles inspissated pus. Sometimes minute purulent foci or abscesses of considerable size may be observed, but these are rare, except in the condition of aspiration-pneumonia.

Whilst the lung is usually crepitant throughout, and may float in water, the inflamed areas, when removed, sink, owing to their airless condition.

Symptoms.—In the *primary* form the onset is sudden. As the disease occurs most frequently in infants, there is often no history of obvious exposure, and the child has been in its usual health. The first indication of illness most commonly is a smart convulsion, which so frequently takes the place of the initial rigor common in adult pyrexial conditions. The pulse-rate and temperature rise rapidly; cough, dyspnœa, and cyanosis soon develop; and the clinical picture within twelve hours becomes practically identical with that of acute lobar or croupous pneumonia. The illness passes off as suddenly or abruptly as it commenced, the temperature rapidly falling to normal with the usual accompaniments of a speedy crisis; and not unfrequently the physician is unable to satisfy himself whether the attack was of the true lobar or of the lobular type, the physical signs being almost identical, and there being no sputum in the case of the infant to assist in the diagnosis. The physical signs will be referred to presently.

The *secondary* form is much more common. The child has been suffering from symptoms of bronchial catarrh for a few days, or it is passing through, or has passed successfully through, an attack of measles, whooping-cough, rickets, scarlatina, or typhoid fever, when an increase in the fever or the accession of a febrile temperature, with cough and quickness of breathing, will indicate the advent of a capillary bronchitis rapidly passing into and involving the air-cells and lobules. The temperature is seldom very high, rarely as high as in lobar pneumonia; it is very irregular, and may be remittent, or intermittent, frequently falling to the normal in the mornings. The cough is difficult, hard, and painful, and owing to the age of the patient any mucus reaching the pharynx is swallowed.

The breathing soon becomes very rapid and shallow, and may reach sixty or more, the expiration being accompanied by a grunt; the accessory muscles of respiration are brought into vigorous action; the soft parts above the clavicles, the epigastrium, and the lower ribs are sucked in during inspiration. The dyspnœa soon tells upon the blood-condition, and cyanosis, as seen in the bluish colour of the lips and finger-tips and duskiness of the face, becomes evident. The pulse is rapid and compressible. Percussion at this stage generally gives

no dulness because the solidified and collapsed lobules are disseminated; later, when a number of inflamed lobules aggregate, a distinctly dull patch may be demonstrated, and several such patches may be discovered over the chest-surface, with resonant lung between. They may change their positions as the disease advances; it is not uncommon to find them symmetrical, and sometimes as they clear up in one region they become evident in a corresponding site on the opposite side, or in some other part of the same lung.

Auscultation reveals the presence of scattered consonating or crackling râles, which may approach in fineness to those heard in the earlier stages of lobar pneumonia; rhonchi are seldom present. Where an aggregation of solidified lobules is appreciable by the dulness on percussion, there may be increase in the vocal fremitus, and the stethoscope may reveal over the dull patch tubular breathing and bronchophony when the child cries or coughs.

As the difficulty of breathing increases from the extension of the disease to other lobules, and from still further collapse of air-cells, the cyanosis deepens and asphyxia supervenes, obliterating cough and dilating the right heart; the hot, dry skin becomes cool, and is bathed in clammy perspiration; the pulse gets more frequent and weak; delirium, drowsiness, and stupor supervene, and death from cardiac failure or suffocation occurs, preceded by convulsions, sometimes before the end of the third day. In more fortunate instances a gradual improvement in all the symptoms sets in slowly, and recovery steadily follows in two or three weeks.

In the aged the symptoms differ but in degree from those in the child; fever, dyspnœa, and gradual signs of suffocation generally supervene upon an attack of ordinary bronchial catarrh, which cause rapid prostration, with brown, dry tongue, great cardiac weakness, muttering delirium, drowsiness, and coma.

Diagnosis.—The most probable mistake is to regard the case as one of *lobar pneumonia*, but this disease is very infrequent in infancy; it begins very suddenly and terminates in crisis almost as suddenly; the fever is higher and approaches the continuous type, the remissions between evening and morning temperature being only slightly marked; the local physical signs are lobar in their extent, and the disease runs a shorter course. It must, however, be remembered that primary broncho-pneumonia begins smartly and may end suddenly, and these cases are very difficult of differentiation; but they are seldom, if ever, strictly *unilateral*. This tendency of true broncho-pneumonia to invade both lungs is the chief help in distinguishing it in those cases where an aggregation of inflamed lobules may give signs almost identical with those found in the lobar variety; but a careful examination of the

opposite lung will generally reveal evidence of lobular implication there. The sputum of lobar pneumonia is almost characteristic, but unfortunately it is seldom seen in children, as it is always swallowed by them. But it may be sometimes recognised in the matter ejected from the stomach.

Acute bronchitis of the smaller bronchi in the aged may be differentiated if the signs of tubal inflammation remain pure, as they generally do in the adult—viz. sonorous and sibilant rhonchi, with non-consonating râles, absence of dulness and of marked fever, with some cyanosis ; but when increase of fever and dyspnœa, with shifting patches of dulness, appear, and consonating râles or crepitations and bronchial breathing are heard over them, the diagnosis may be made that the so-called capillary bronchitis has extended into the air-cells, and that the disease has passed into broncho-pneumonia.

Acute tuberculosis closely resembles broncho-pneumonia, and the diagnosis can only be made after a painstaking scrutiny of the history and a daily watching of the physical signs and fever. There is only one sign which can be relied upon, and this is the presence of tubercle bacilli in the sputum ; and as this latter is so difficult to procure, an emetic may be administered with the view of obtaining a sample. Both diseases may occur together, and tubercle is sometimes a sequela.

Pleuritis and *empyema* may be excluded by the bilateral character of the signs and by the presence of consonating râles and bronchial breathing over the duller base ; but sometimes the case cannot be cleared up without the hypodermic needle being used as an aspirator.

The affection will be again referred to under 'Pneumonia,' where its relationship to the croupous variety of the disease will be discussed.

Prognosis.—This is grave in the youngest subjects and in the aged, and in both death may occur by the third day. It is much brighter in primary cases than in patients who are suffering from measles, whooping-cough, and typhoid fever, and the gravity will be influenced by the stage and severity of the primary disease in these cases.

The gravity of the case generally depends upon the number of lobules involved and the amount of pulmonary collapse, and these can be inferred from the cyanosis and extent of the dyspnœa and frequency of the pulse. The mortality is always higher than in acute lobar pneumonia, and the prognosis should always be of the gravest in the broncho-pneumonia caused by aspiration of septic material, or of the contents of bronchiectatic and phthisical cavities.

Treatment.—This in the main corresponds with the use of such agents as are found valuable in acute bronchitis and acute pneumonia. The child should be confined to bed or be

permitted to remain in the nurse's lap enveloped in a light blanket; the air of the room should be kept warm and moist, a temperature of 60°–65° F. being desirable; the ordinary bronchitis-kettle should be kept boiling on the fire in the grate, as the various forms of gas-burners and lamps for this purpose are highly objectionable, owing to the vitiation of the atmosphere which they produce.

Liquid diet, chiefly milk, should be given at short intervals, and beef-juice with a little stimulant reserved for the later stages when there are signs of exhaustion.

Pain and pulmonary distress are to be relieved by mild counter-irritation, afterwards followed by a jacket-poultice; but where the application of this causes much excitement to the little patient, it is wiser to be content with a thick layer of absorbent wool under thin mackintosh. Cold or ice locally is used by some physicians; but the writer has never had the courage to persist in this remedy. Cold or tepid sponging is unobjectionable when the temperature is high; rarely will the pack be necessary. The routine administration of expectorants must be conducted with caution, and the physician is wise who invariably discards opium in all forms. The best expectorant will be sal-volatile, of which an infant one year old may get 3–5 mins. well diluted with syrup; it should be combined with 5–10 mins. ipecac. wine, and as much Mindererus spirit. This combination may be pushed every fifteen minutes when an emetic effect is desired; otherwise, every two hours will be sufficient. Very acute cases should receive a smart purge, followed by 5-min. doses of antimonial wine every hour till the skin begins to act freely, and an impression be made upon the temperature. The best form of stimulant is wine-whey, made by adding a glass of sherry to a pint of hot milk. Oxygen should be given, diluted well with air, when cyanosis threatens to become marked, and strychnine will be indicated when the cardiac muscle and respiration begin to flag; even artificial respiration may be demanded. The after-treatment must be conducted upon general principles, and in the slow forms following measles and fever, change of air and forced feeding may be indicated in order to guard against the advent of phthisis, as soon as the prominent bronchial symptoms have subsided.

CAISSON-DISEASE

This, also known as Compressed-air Disease and Diver's Paralysis, is a condition characterised by pains and paralysis which come on after sudden removal from exposure to air at a pressure of three or more atmospheres.

Symptoms.—The diver or worker in a caisson or diving-bell, after coming to the surface where the atmospheric

pressure is normal, is liable to be seized with pains of a most excruciating neuralgic character in the limbs or trunk, but especially in the joints and muscles of the lower extremity and back, though every part of the body may be involved. These agonising pains may come on immediately, or be deferred for a few hours, and they are liable also to be followed by, or accompanied with, paraplegia and cerebral disturbances, as vertigo, headache, double vision, vomiting, &c. Sometimes the cerebral symptoms rapidly pass into coma, and death may result in half an hour, or be delayed for several days. The intensity of the symptoms vary with the pressure, the duration of the exposure, and the rapidity of the transition from the higher to the normal pressure ; and it is always worse in stout subjects. The paralysis generally is associated with loss of sensation, and in the great majority of cases it passes off after some hours or days, but occasionally it may last for weeks, or even months.

Morbid Anatomy and Pathology.—In those cases where *post-mortem* examinations were obtained, there were found congestion of the spinal cord, hæmorrhages, and sometimes myelitis, and congestion of the brain and internal organs. These changes have been supposed to be due to the mechanical injury caused in the spinal cord by the escape of bubbles of nitrogen gas which, under the high atmospheric pressure, had been previously absorbed by the blood.

Whatever differences of opinion may exist about the mechanism of the symptoms and *post-mortem* changes, there can be no doubt that the condition is caused by the suddenness of the change from the higher to the normal atmospheric pressure. When this is properly graduated, the symptoms rarely appear ; and when they do, they are immediately dispelled by returning the patient at once to the higher pressure. When an intermediate chamber or lock is established where the caisson-worker is slowly subjected to a gradual diminution of pressure before returning to the surface, the symptoms are entirely prevented. The erection of a suitable high-pressure air-chamber, as suggested by Andrew Smith, into which the patient can be conducted immediately upon coming to the surface, and where the pressure can be slowly and gradually reduced, has led to the total prevention of the illness ; the same result is obtainable by a longer sojourn in the intermediate or lock chamber before reaching the surface. The disease very rarely terminates in death.

Treatment.—The pain may be so intense as to demand large hypodermic doses of morphia, and the muscular weakness or paralysis, when these persist, must be treated upon general principles by massage, strychnine, counter-irritation, and electricity, as in cases of chronic inflammation of the cord.

CANCER

Little need be said under this heading, since cancer of the various organs is referred to under the articles dealing with the diseases of each portion of the body. In no other department of medicine has such laborious research-work been so unceasingly pursued during the last decade as in the efforts to clear up the etiology of this dreaded disease.

The parasitic theory of the causation of cancer has many adherents; but notwithstanding the enormous variety of organisms which have from time to time been figured as the parasites of cancer, it must be admitted that if any causal microbe exists it still awaits discovery. '*Plimmer's bodies*,' whatever be their nature, have now been accepted to be non-parasitic. They are found as round or oval clear vesicular bodies situated in the cell-protoplasm and separated from its periphery by a clear space, each being surrounded by a definite limiting membrane, and containing in the centre a dark spot. They are considered by Greenough to be vacuoles in the cell-protoplasm, consisting of a material secreted by the cell; other observers regard them as the results of a degenerative change or of abnormal methods of mitosis, &c. Similar bodies have been found in normal reproductive tissue in the cells in various stages of their multiplication.

Doyen's *micrococcus neoformans* has suffered a like fate, though its very frequent presence in malignant growths has been indisputably proven by numerous workers in this domain. Its presence in 80 per cent. of all undoubtedly cancerous growths is accepted by Copeman as evidence that it must be regarded as something more than a casual concomitant; and he is inclined to accept it as the causal agent of a terminal infection exerting a somewhat similar influence to that which has been attributed to the *diplococcus lenticularis* in conjunction with the true specific *trypanosoma Gambiense* in sleeping sickness.

The *micrococcus neoformans* is polymorphic, occurring as a staphylococcus, a diplococcus, or in groups of several units. It can be cultivated in bouillon and stains by Gram's method. In its morphology, and the characters of its growth on gelatin and agar, it differs from all other allied organisms, and Wright has shown that it differs from staphylococci in several fundamental particulars. It bears a very close resemblance to Welch's comparatively harmless skin-parasite, *S. epidermidis albus*. Doyen claimed that injections of pure cultures of his organism caused lesions analogous to cancerous growths, but in this vital detail his researches have not been considered as established. The discovery and isolation of the organism caused great interest during the past few years from the

publication of an announcement that a serum, prepared from the blood of horses, injected by cultures of the *m. neoformans*, afforded a curative agent in the treatment of cancer. The hopes raised by this premature statement unfortunately were blasted by the utter failure of the agent. A similar failure has followed the plan of employing a vaccine consisting of measured doses of sterilised cultures of the micrococcus, as carried out by Wright's methods.

The researches of Moran, Jensen, Boral, and numerous others in the spontaneous and experimental forms of cancer in mice have aroused the keenest interest and excited hopes that the many hitherto insoluble problems associated with the etiology of cancer may find a solution by the study of the comparative pathology of the disease. Cancer has now been found to occur in numerous animals, and as the result of the elaborate researches of late years it is accepted as practically proven that the disease is distributed throughout the entire vertebrata, with the exception of the reptilia, amongst whom no case has yet been recorded.

Jensen, of Copenhagen, following Moran who is stated to have first produced experimental cancer in mice in 1891, has carried out this method in a large number of cases, and the work has been actively pursued by Bashford, Murray, and Cramer in the Imperial Cancer Research Laboratories.

A disease closely resembling human carcinoma is frequently found as occurring spontaneously in the mouse, and is easily inoculable, producing tumours in other mice, which grow to comparatively enormous dimensions. The original tumour-tissue from a spontaneous case of the disease obtained by Jensen is still being employed, after having been passed through countless numbers of the animals, and its intensity is such that in the hands of some experimentalists the rate of success in the transplantations has sometimes reached to 100 per cent. Handley mentions that in France secondary tumours were produced by inoculating a portion of a primary mouse-tumour into two human subjects by an observer who remains anonymous ; though Copeman, in his recent 'Guthrie Lecture on Cancer,' states as his belief that there is no instance on record of the successful transmission of cancer from man to the lower animals, or from one animal to another of a different species.

In these transplantations of mice-cancer, the new cells, after inoculation, increase and multiply enormously, the normal cells of the tissues of the host taking no part in the process, though the stroma of the new-growth is provided by the tissues of the inoculated animal ; the stroma-tissue, inoculated with the cellular elements of the growth, perishes speedily.

Thus, as pointed out by Copeman, it has been demonstrated step by step that the inoculation-tumours are made up of the

genealogical descendants of the cells experimentally introduced. If the spontaneous mouse-cancer be accepted as identical with human carcinoma (an assumption, however, which is disputed by Barlow and Hansemann), the researches would seem to support the older view of Cohnheim, that carcinoma originates in embryonic cells, whose development having been arrested, their remnants remain dormant amongst the normal tissue-elements, till, at the cancer age, they begin to increase and multiply.

The former view, still adhered to by many pathologists, that the origin of carcinomatous growths is explained by the theory of some degenerative or other change occurring in the normal cells of the tissue or organ which becomes the seat of the neoplasm, does not appear to receive much support from the results of these experimental inoculations. As pointed out by Handley, the great difficulty in accepting the parasitic origin of cancer is that 'the apparently limitless proliferative power of malignant cells has no analogy to the limited reactive cell-proliferation which results from invasion of the tissues by any known parasite.' This prodigious proliferative power of the cells of mouse-cancer causes apparently no cachexia, though the secondary tumour may become within a week much greater than the weight of the original body of the host ; this has led some observers to accept the view that each cancer-cell should be regarded as a parasitic protozoon.

A very important result has been obtained in some cases of inoculation of mice. The original adenoid type of tumour-tissue which has been made to pass through succeeding generations of mice becomes gradually transformed into a true sarcoma which is capable of reproducing itself through almost an indefinite number of generations.

Occasionally spontaneous recovery has followed the inoculations, in which cases the convalescent mice were found to be immunised for a period of about six months, and Clews was able to immunise other mice by the injection of the blood of these. This latter fact affords some reason to hope that a serum-therapy may still be possible. Up to the present, the only hope in the treatment of the disease lies in early and complete removal of the neoplasm by surgical means ; though some amelioration is sometimes obtainable by injections of Coley's fluid, by the use of the X-rays, and by the trypsin treatment.

The number of deaths from cancer registered of late years has been steadily increasing, and the subject has caused considerable interest in both lay and professional circles. There are good reasons for regarding the increase as being mainly due to the increase in the average of life, which has been steadily rising during the last half-century owing to improved hygiene. The proportion of individuals surviving to the cancer

age is larger than formerly, and when the cancer mortality is only compared with the total mortality it must be borne in mind, as pointed out by Handley, that the recent diminution in infantile mortality and in deaths from tuberculosis, diphtheria, &c., will produce an apparent increase in the mortality from cancer. Another significant fact is that the recent increase in the registered cancer-deaths is mainly due to the greater number of deaths recorded as due to cancer of the internal organs, which were formerly not diagnosed, owing to the imperfections of the older methods of diagnosis. The frequency with which exploratory incisions and abdominal operations are being performed bring to light many cases of cancer which formerly were regarded as examples of other diseased actions.

Copeman states that amongst those above the age of thirty-five now living, the chance of ultimately dying of cancer is one in eight for women and one in twelve for men. It is, moreover, generally accepted that the disease is showing a tendency to appear at a somewhat earlier stage of life than in past years, and that the apparent or real increase is chiefly amongst males. The different varieties of cancer, as scirrhus, encephaloid, adenoid, epithelial, colloid, and alveolar, are referred to in the descriptions of the disease as it affects the different organs and tissues.

CANCRUM ORIS

This is a form of stomatitis, known also as Water-canker, Noma, or Gangrenous Stomatitis.

Etiology and Pathology.—The affection occurs in debilitated children between two and five years old ; more frequently girls are affected, and the majority of the cases occur in ill-fed or neglected patients recovering from measles, scarlatina, and typhoid fever. No specific micro-organism has yet been conclusively demonstrated, though numbers have been described. Lingard's thread-like bacillus has been discovered in the tissues surrounding the gangrenous area, and Bishop Ryan describes an organism which he has isolated, and which resembles an attenuated form of the diphtheritic bacillus.

Symptoms.—An indurated spot or a bulla forms on the mucous membrane of the inside of the cheek, which rapidly ulcerates, and all the tissues lying between the mucous membrane on the inside and the skin on the outside of the cheek become rapidly gangrenous. Brawny induration surrounds the necrosing area, and in the centre of this, after several hours, the red skin becomes black, a bleb forms, and perforation may be noticed. The gangrene speedily attacks the gums and jaws ; the teeth loosen and drop out ; and if the

patient lives, in less than a week the bones of the face and orbit may be destroyed by the necrotic process. There is little or no hæmorrhage, and pain is slight or absent, but the prostration is extreme, though fever is not generally high ; low muttering delirium and diarrhœa soon supervene, and often bronchopneumonia, before the patient sinks from the exhaustion caused by the absorption of the foul putrefactive products of the gangrenous action.

Prognosis.—A small number, probably not more than one-fifth or one-sixth of those affected, survive, and many of these are permanently marked by considerable facial deformity, not less than 80 per cent. of the cases dying even when treatment is prompt.

Treatment.—The only hope of arresting the gangrenous process is by the immediate removal of the sloughing tissues. Should the case be seen in its very early stage, the ulcer should be thoroughly scraped and fuming nitric acid freely applied under an anæsthetic to the diseased mucous surface. Once the necrotic process has got fairly started the acid alone is useless, the knife or cautery must be freely carried outside the gangrenous zone, and all diseased tissue removed. The parts must be kept saturated by antiseptic solutions. Constitutional treatment, by over-feeding with strong soups, beef-juice, and rectal alimentation, is of vital importance.

CARBUNCLE. (See under Boils, p. 109.)

CATALEPSY

In the majority of instances this is but a manifestation or symptom of hysteria ; but the condition is also sometimes found where there is no reason to suspect hysteria—*e.g.* in meningitis, and especially in insanity of the melancholic type. It is a rare affection, generally occurring in young adult females after some violent excitement or emotion preceded by nervous exhaustion.

The chief phenomenon is the strange condition of the muscles, which comes on suddenly with more or less loss of consciousness ; the limbs become rigid and assume the odd attitudes so often seen in hypnotic demonstrations. The state of the muscles, though rigid at the onset, generally passes into one of plasticity, when the limb can be put into any statuesque position by the physician, as if it was formed of soft wax or modeller's clay, and in this position it remains for a long time before the wearied muscles yield to the force of gravity ; hence the older writers called the condition '*flexibilitas cerea*.' Sensation may be entirely lost, and reflex movements abolished,

though all sorts of modifications of the mental and physical state may be noticed in hysterical cases. The duration of the attack varies likewise ; but it may last for several hours, and come on in paroxysms occurring during many days.

The pathology of the affection is attributed by Gowers to a deficiency of control in the motor cortical centres permitting their over-action, the spinal mechanism being secondary and subordinate ; and this view is supported by a study of the manifestations of hypnotism. It sometimes is associated with the condition known as Trance.

Diagnosis.—The recognition of the affection is easy in its typical form, but various cataleptoid conditions may cause difficulty ; the contractions of limbs, seen in *hysteria*, are to be differentiated by the impossibility of overcoming the rigidity which is always present.

Malingering may be detected by applying a weight to the rigid limb. In catalepsy this soon overcomes the rigidity ; but in feigned cases the condition persists much longer, and if the cord supporting the weight be suddenly cut, the limb bounces upwards.

Prognosis.—The outlook depends upon the condition to which the cataleptic rigidity is secondary ; thus in meningitis and melancholia it is more or less grave in proportion to the severity of the other brain-symptoms. In hysteria the prognosis is always good, though the progress may be slow, and the liability to relapses very great.

Treatment.—Some mild form of shock or stimulation of the sensory nerves by a cold douche or faradism may be tried. The vapour of ammonia or acetic acid to the nostrils, and the evacuation of the stomach-contents by a hypodermic of apomorphine, sometimes cut short the attack. In melancholic and meningeal cases, heroic douching and electrical shocks are contra-indicated. In the interval between the attacks the alteration of the patient's environment and the recognised anti-hysterical treatments should be persisted in.

CEREBELLUM, DISEASES OF

The functions of the cerebellum are still to a large degree matter of speculation, and though this portion of the brain is liable to the same diseases as the cerebrum, pons, and medulla, the means of localising or diagnosing the various diseased conditions are surrounded with great difficulties. The most characteristic symptom of cerebellar disease is the gait, which is generally described as 'ataxic,' though there is no real incoördination of muscular movement in the so-called cerebellar ataxia. The gait is very unsteady, the patient swaying from

side to side, reeling or staggering like a drunken man, and his progress in walking is distinctly zigzag.

This gait is easily distinguished from that characteristic of locomotor ataxia by the above features, as shown by the fact that, though the patient may stand with difficulty, the closing of his eyes makes no practical difference to him, and if he lies down he can execute perfectly the most delicately coördinated movements.

Other symptoms frequently caused by cerebellar disease are occipital headache, vomiting, vertigo, and sometimes paralysis, but these are in no way distinctive.

Abscess of the cerebellum may sometimes be recognised by the drunken gait, pain over the occiput and nucha, retraction of the head, nystagmus, vertigo, and optic neuritis. The occipital headache is very severe, and the vomiting persistent. Most of the cases occur from middle-ear disease, where the mastoid or where the external ear is affected ; whilst tympanic disease is more likely to lead to temporo-sphenoidal abscess. Macewen finds that in abscess of the cerebellum he can elicit a tympanitic note, which aids in the diagnosis.

The prognosis is fairly good where the absence of symptoms exclude pyæmic cases, and where operative treatment is resorted to early. The rules for the treatment of abscesses elsewhere apply to those of the cerebellum. The contents should be speedily evacuated, and the operation of trephining and passing a probe or trocar into the abscess-cavity has given most excellent results, recovery following in the majority of cases.

Hæmorrhage into the cerebellum is comparatively rare, and often there are no localising symptoms, the patient being suddenly seized with severe vomiting and occipital headache, rapidly followed by coma and general paralysis. Sometimes in mild cases hemiplegia can be made out, but more frequently it is absent. When present it is probably due to pressure upon the motor tract ; if this involves the medulla the paralysis will be upon the same side as the hæmorrhage, whilst if the pressure interferes with the pons it will be on the opposite side. Death may be very rapid when the hæmorrhage invades the fourth ventricle, causing respiratory failure.

Where the middle peduncle is involved, rotatory movements often occur. After the disappearance of the urgent symptoms recovery may set in, and the characteristic drunken gait may remain as the only evidence of the lesion. If with this there is weakness of the limbs on one side of the body and exaggeration of knee-jerk on the same side, it is almost certain that there has been a hæmorrhage into the corresponding hemisphere of the cerebellum. Priapism has been frequently noted as a prominent symptom in hæmorrhage into the middle

lobe, and this is explained by the irritation which results to the posterior surface of the medulla and pons.

Tumours of the cerebellum are of the same nature as cerebral tumours; they may lead to no symptoms during life, especially if the middle lobe be not involved. One hemisphere has been repeatedly found destroyed by a growth, and no symptoms resulted; but if the tumour be large enough to press upon the pyramids or the pons, pressure-symptoms show themselves.

As mentioned upon the previous page, the most characteristic sign of cerebellar disease is the gait; this is present when the middle lobe is affected by tumour, and is of the greatest value in diagnosis; vertigo accompanies it. When this symptom (vertigo) is severe and persistent in the case of an intracranial growth, the site may be safely localised in the middle lobe of the cerebellum. Vomiting, severe headache, tremors of the hands and arms like those seen in disseminated sclerosis, optic neuritis, paresis of the limbs on one side of the body or of the trunk-muscles, nystagmus, retraction of the head with neuralgic pains over the occiput and nucha, and chronic hydrocephalus from blocking of the veins of Galen, and ventricular distension, may be present. When the tumour is situated in the superior, middle or inferior peduncle, forced movements or actual rotation round the vertical axis, with sometimes deviation of the optic axes, may be observed.

Prognosis.—In the case of cerebellar tumour the prognosis is similar to that in cerebral tumour; but it must be remembered that very extensive disease may exist without any danger to life. This is seen in the many reported cases of atrophy of the cerebellum, in some of which the whole organ was found completely atrophied. Tumours, however, sometimes cause death in a few months after their first symptoms are noticed; but they may remain stationary for a long period, and then cause death with startling rapidity from paralysis of respiration. The prognosis is fairly good in syphilitic cases where the vessels are not extensively diseased.

Treatment.—This is identical with that to be pursued in other cases of intracranial tumour. Mercury and iodides are indicated in syphilitic cases, and the use of such analgesics as will relieve pain and symptoms as they occur. Owing to the position of the cerebellar tumour, there is little hope of surgical removal, especially when the middle lobe is the site.

CEREBRAL ABSCESS

This condition is also known as Suppurative Encephalitis.

Etiology and Pathology.—The disease is three times more common in males, and occurs oftenest between the ages of ten and thirty years. Primary cerebral abscess probably never

occurs ; it may be regarded as always secondary to (1) *Injury*, such as blows causing fracture of the skull, or scalp-wounds leading down to the bone ; though suppuration may occur without any breach of the cutaneous surface or without necrosis. (2) *Extension from disease of the middle ear or mastoid cells.* (3) *Distant septic infection*, either as a part of a general pyæmia (which, however, is rare) or more commonly by septic infection carried from the lung, or from an empyema, in which cases the cerebral abscesses are often multiple.

By far the most frequent cause of cerebral suppuration is that which arises from extension of chronic suppurative disease of the middle ear and mastoid cells to the cerebral tissue. Generally, but not always, there is necrosis of bone. The extension may be direct through the thin plate of bone in the middle fossa which forms the roof of the tympanum, in contact with the temporo-sphenoidal lobe, or through the roof of the mastoid antrum lying underneath the cerebellar hemisphere in the posterior fossa. The extension may occur by the septic foci finding their way into the sinuses, or by a slower invasion of the membranes, beginning in the dura and extending to the pia after involving the arachnoid. Most probably the infection is carried by the lymph-canals which surround the cerebral vessels. Even when the membranes are found inflamed, the abscess may be deep and separated from the pia by a layer of apparently healthy cerebral matter, and sometimes the membranes are normal over a deep abscess.

The abscess is generally solitary. The cerebrum is much more frequently involved than the cerebellum ; this latter is affected by extension from the mastoid, whilst the temporo-sphenoidal lobe suffers from the proximity of the tympanic roof. The abscess may be of any size : often it is the size of a bantam's egg ; a capsule may or may not surround it ; its contents are green, with an acid reaction, and are sometimes fetid. All forms of pyogenic bacteria have been found in the pus, the most common being *streptococcus pyogenes* and *staphylococcus pyogenes aureus*. It may burst into the ventricles or on the surface of the brain, causing purulent meningitis, or more commonly may cause death without discharging, or it may remain quiescent for a long period.

Symptoms.—The symptoms are very variable in their onset, progress, and termination ; generally the suppurative process in the cerebral tissue begins treacherously after many years of chronic ear-disease, during which otorrhœa has been more or less a constant symptom. Occasionally, however, cerebral abscess supervenes upon a recent acute attack of otitis complicating scarlatina or measles, and rarely its first symptoms may be traced to an extension from chronic or acute disease of the nasal fossa

Some cases show acute onset and progress, and terminate fatally within a fortnight; these are generally of traumatic origin, and the chief symptoms are those of meningitis—*i.e.* fever, intense headache, rigors, and vomiting, rapidly passing into delirium, convulsions, paralysis, and coma. More frequently the course is slow in ear-cases; and after an onset, in which the initial meningeal symptoms are so trivial as to be overlooked, the disease becomes entirely latent for weeks or months, during which time there may be no sign or symptom that anything is wrong. This is especially the case when the abscess, which may be a large one, is situated in a capsule in the frontal or temporo-sphenoidal lobes. This 'latent' period ultimately terminates with the bursting of the abscess or with the extension of the original inflammatory process to the surrounding healthy cerebral tissue, when the symptoms of what Gowers calls the 'terminal' stage may develop suddenly; these are practically identical with the symptoms which are prominent in the acute traumatic cases from the start. The most typical examples of cerebral abscess following ear-disease are those in which the so-called latent stage is never completely latent, but after an ill-defined, and often overlooked, beginning, with mild irritative meningeal symptoms and earache, these become more or less chronic, and finally the headache deepens gradually, and ultimately becomes very severe; vomiting persists or develops; delirium and convulsions appear, followed by hemiplegia; the fever rises and rigors come on, stupor and coma closing the scene. The pulse is very variable, sometimes being slow at first and quick towards the end, but sometimes the reverse happens; optic neuritis and paralysis of the ocular and facial muscles may be present, the latter from bone-disease. In many cases there is no rise of temperature, probably owing to the absence of sinus phlebitis or meningitis.

The localisation of the abscess may be impossible when the frontal and temporo-sphenoidal lobes are involved. Hemiplegia when present is never complete; it may ensue where a large temporal-lobe abscess presses upon the motor tract, when the face and arm may be paralysed, and there may be agraphia, aphasia, and word-blindness. When temporo-sphenoidal abscesses are large, paralysis of the third nerve on the same side is common, and the face may be involved on the opposite side. Considerable dulness and intellectual failure, with aphasia and weakness of the neck-muscles of the opposite side, may mark an abscess in the frontal lobe. The characteristics of an abscess in the cerebellum have been mentioned upon page 162; they are the drunken gait, occipital pain, vertigo, retraction of the head, and incessant vomiting.

Diagnosis.—In chronic cases with a long latent period, where the pus is encapsuled in the silent regions of the cerebrum, diagnosis may be impossible. In the cases caused by injury there may be an interval of very many months of apparent health between the blow and the terminal stage; but in those where fever, delirium, stupor, &c., supervene soon after an injury the diagnosis is generally clear from the commencement.

The point of most importance in the diagnosis of cerebral abscess is the recognition of the ear-disease which is the most common cause; in the absence of evidence of it, or of nose-disease, or of the septic processes in distant parts which may determine a cerebral suppuration, the diagnosis may be doubted. In other words, if no cause can be found the symptoms must be regarded as probably those of *meningitis* or *tumour*.

Meningitis of the suppurative type is likely to have a high temperature, and its onset is more sudden; tubercular disease elsewhere may be found in tubercular meningitis, and aphasia and paralysis of limbs are not commonly present, though meningitis is liable to affect the cranial nerves.

Tumour generally produces a more severe and intense headache (except in cerebellar abscess); optic neuritis is more strikingly marked than in cerebral abscess, and the localising symptoms indicate that the site of the tumour is not that generally seen in abscess. The temperature in tumour is generally normal; and though it is often so in abscess in the acute cases, in the early stage of the chronic cases of suppuration it is elevated and irregular. The progress of a tumour is, upon the whole, slower than that of a chronic cerebral abscess.

Sometimes the mistake is made of diagnosing a cerebral abscess when the symptoms are those of ear-disease without any cerebral extension or suppuration, as the pain in ear-disease may take on the character of a severe headache, with vertigo and vomiting. The pulse may assist. In these cases it is always quickened in ear-pain, and if marked slowness exists, with signs of paralysis, stupor, or mental failure, abscess may be certainly regarded as having supervened.

Mastoid abscess confined to the cells, without extension to the cerebral tissue, may produce sinus-thrombosis and double optic neuritis.

Prognosis.—This is always most serious and generally of the gravest, since, unless the case is one suitable for operative treatment, death is certain. With improvements in the technique of the operation the mortality is steadily diminishing, as Macewen's figures demonstrate. It is, however, always high in cerebellar operations, owing to the difficulties in reaching the abscess; and the cerebellar site is twice as frequent as the temporo-sphenoidal in ear cases.

Treatment.—The question of operative interference must be immediately considered as soon as the symptoms warrant a diagnosis. In cases of injury, trephining over the spot should be at once resorted to.

In ear-cases, because of the frequent absence of localising symptoms, as just described, owing to the site of the abscess being deep in the silent temporo-sphenoidal lobe, the indications for trephining are not so obvious; but they are no less imperative. A small opening should be made with the trephine, and the temporo-sphenoidal and lateral cerebellar lobes explored by a fine trocar or probe directed through the cerebral tissue to the suspected site of the abscess, under stringent aseptic precautions.

The treatment in cases outside the sphere of surgery should be conducted upon the principles indicated for the relief of headache, &c., as in meningitis—*i.e.* the ice-cap, counter-irritation, antipyrin, &c.

CEREBRAL EMBOLISM AND THROMBOSIS (ARTERIAL) AND BRAIN-SOFTENING

Etiology and Pathology.—An embolus which causes occlusion of a cerebral artery generally comes from a fibrinous growth on an old diseased mitral valve, most commonly in the case of mitral stenosis, or it may originate in the detachment of a portion of the fibrinous clot which often exists in the dilated auricular appendix in this form of mitral disease, or in failing compensation. It may, however, be a portion of detached fibrin from a regurgitant aortic lesion, or from an atheromatous roughening on the inner surface of the aorta or large vessel in the neck, or from an aneurism. Sometimes it is a small clot, or a portion of one, which has formed in the left auricle or ventricle owing to the increased coagulability of the blood which occurs towards the end of pregnancy or during lactation; ulcerative endocarditis may also produce the condition. It is extremely unlikely that any particles set loose in the veins would be able to pass through the pulmonary capillaries and reach the cerebral vessels in such a way as to produce the phenomenon of cerebral embolism; but the writer has several times witnessed very sudden death in the rabbit during the injection of an emulsion of spinal cord into the ear-vein, which appeared to be produced by the blocking-up of the cerebral capillaries with organic material which had traversed the larger pulmonary capillaries of the animal without producing lung-symptoms. In old empyemata and in phthisis, foci sometimes find their way into the pulmonary veins and cause blocking in the small vessels of the brain.

Owing to the peculiarity of the distribution of the arteries in the brain, the blocking of a branch leads generally to a rapid destruction of the area supplied by it, because there is little anastomotic provision, and in the case of the vessels supplying the great central ganglia there is practically none.

The artery most frequently affected is the middle cerebral or one of its branches, and the left is somewhat more frequently occluded than the right. The portion of cerebral tissue formerly nourished by the plugged vessel rapidly softens, so that in forty-eight hours it shows marked changes in its consistency owing to the breaking-up of the nerve-elements; free fat-granules, shreds of nerve-fibres and red blood-cells are found in it. In proportion to the amount of venous engorgement the condition of the so-called *red softening* becomes evident, and this is most frequently observed in the grey substance owing to its greater vascularity; more or less inflammatory action and a considerable amount of œdema are also observable. *Yellow softening* follows owing to degenerative changes in the blood-elements; and when a large vessel is occluded extensive yellow softening in the form of the so-called *plaques jaunes* may be found scattered throughout the convolutions in the affected area. *White softening* is chiefly seen in the white matter, and it may proceed to the extent of fluidity.

There is always some inflammatory action around the damaged tissue which tends to deepen the clinical symptoms, but this with the local œdema may soon subside. Ultimately the softened area may become a cyst or be entirely absorbed, leaving nothing but a cicatrix. The occluded vessel may dilate upon the cardiac side of the plugging, and a small aneurism may develop upon it; but this is most likely to occur when the embolus is septic.

Symptoms.—The symptoms of cerebral arterial embolism will vary with the region of the brain affected; in the main they resemble, and are often identical with, the symptoms of cerebral hæmorrhage. When a large area is suddenly deprived of its blood-supply there may be all the signs of a severe apoplectic seizure, with unconsciousness; and when the convolutions are involved, convulsions may be present. The onset is always sudden, no premonitory symptoms being noticed; but, as a rule, the coma is not so profound and does not last, though death in some cases rapidly follows embolic plugging of a large vessel without return of consciousness. Paralysis is soon obvious, generally of the hemiplegic type, and the right side of the body is much more frequently affected than the left. Consciousness may be retained throughout, the only interference with it being due to the sensation of intense headache or vertigo. When the occluded vessel is small and the anastomoses numerous, no symptoms may be

produced at all or there may be trifling paralysis, which rapidly passes off.

The nature of the paralysis, which commonly affects one-half of the body, will be further referred to under 'Cerebral Hæmorrhage' and 'Aphasia.'

Thrombosis in a cerebral artery leads to similar changes in the cerebral tissue and to symptoms in the main resembling those following embolism and hæmorrhage.

Etiology and Pathology.—The cause is disease of the coats, either of an atheromatous or tertiary syphilitic character; but the thrombus may grow around an embolus arriving from a distance, or it may possibly take its origin in some obscure blood-condition.

Symptoms.—As a rule, the onset is not so sudden. There are generally premonitory symptoms, as severe headache, giddiness, and numbness or tingling, often confined to the regions which become ultimately paralysed. Mental symptoms, as loss of memory, temporary aphasia, drowsiness, or irritability of temper, may come on hours or weeks before the final seizure; occasionally, however, this may be almost as sudden as in hæmorrhage. Consciousness is often preserved, especially in syphilitic cases, even when hemiplegia and other symptoms are well marked. In senile cases the symptoms are often obscured by the previous mental condition, and in these instances, after partial recovery, relapses are common. The onset is often in two or more stages, especially in syphilitic cases, a slight attack characterised by muscular weakness being followed several days afterwards by severe hemiplegia.

There is little, if any, change in the temperature, but sometimes a slight drop may be noticed immediately after the attack. In basilar occlusion hyperpyrexia is often present in the later stages after a marked initial fall, but in most cases of extensive cerebral plugging there is a well-marked rise owing to secondary inflammation several days after the attack, and delirium and convulsions may usher in this change.

In many instances it is not possible to determine the site of the embolus or thrombus, and in any case the changes will largely depend upon the exact spot in the vessel where the occlusion has occurred and on the peculiarities of the vascular distribution. When plugging involves the trunk of the internal carotid, for example, no results may follow, the vessels in the circle of Willis being able to carry on the circulation by dilatation of the communicating arteries; but when these are absent or small, softening follows. If the obstruction be caused by an embolus at the point of division of the internal carotid, the anterior cerebral should receive its supply from the anterior communicating, when this vessel is normal, and then the region affected would be that supplied by the middle

cerebral artery; but in thrombosis of the internal carotid the clotting generally extends into all its branches. The occlusion of the middle cerebral artery itself is the most common cause of cerebral embolism or thrombosis, and the symptoms will vary considerably according to the number of its branches which escape or which are involved, as will be seen under 'Aphasia.' Basilar occlusion, if complete, produces paralysis of the limbs on both sides of the body, and bulbar symptoms with marked initial drop in the temperature followed by hyperpyrexia, Cheyne-Stokes breathing, and symptoms identical with hæmorrhage into the pons, ending in death within a week.

Vertebral thrombosis or embolism is very variable owing to irregularities in the vascular supply of the medulla; there is transient hemiplegia with bulbar symptoms on the same side.

Plugging of the anterior and posterior cerebrals is comparatively rare, and the only symptom in the former may be loss of smell, and in the latter hemianæsthesia.

Diagnosis.—As between embolism and thrombosis, the symptoms being in many cases identical, the differentiation is difficult. The onset is generally very sudden in embolus; there is very often a history of premonitory symptoms in thrombus. The most important point, however, is the presence or absence of the diseased conditions which lead to each variety of plugging. Thus old valvular disease, or dilatation of the left auricle, or the evidence of emboli in other vessels, or in the spleen, and the presence of the puerperal state, will point to emboli. The presence of extensive atheroma, in old subjects, and the evidence of tertiary syphilis in patients under forty, point to thrombosis. In syphilis the accessible arteries will be found stiffened, and nearly always a history of cerebral symptoms threatening an attack can be obtained, the symptoms, coming on slowly, gradually deepen or pass off, making way for a more complete and severe attack; thus in syphilitic thrombosis the symptoms are either ingravescent or the seizure may have two or more stages separated by a few hours or days of apparent recovery, and consciousness is generally retained. The diagnosis of arterial occlusion from cases of cerebral hæmorrhage will be found under 'Cerebral Hæmorrhage.'

Prognosis.—This will depend upon the size of the vessel occluded and the importance of the parts supplied by it. It is worse in thrombosis, and always more serious when a vessel at the base of the brain is affected. Thus basilar occlusion is almost always fatal, and the prognosis is nearly as bad when the internal carotid is thrombosed; when both middle cerebral trunks are involved death is certain. The intensity of the coma or apoplectic condition is always grave,

but recovery may follow after a moderate degree of stupor has lasted several days ; Cheyne-Stokes respiration nearly always indicates a fatal termination. First attacks often end in at least partial recovery. Gowers has pointed out a common fallacy regarding the prognosis in syphilitic cases. As regards the paralysis and softening, these processes cannot be altered, as the occlusion cannot be removed ; the prognosis is therefore the same as if the case were non-specific, but treatment is of the greatest value in preventing the plugging extending to other vessels afterwards. The prognosis, generally speaking, is more hopeful in the young, but it deepens where there are evidences that the embolus contains septic organisms.

Treatment.—The initial apoplexy is to be treated by absolute rest and slight elevation of the head ; the patient should be kept at rest upon the spot where his seizure has occurred when this is possible. If the diagnosis is certain, blood-letting must not be thought of, as this would certainly increase the tendency to thrombosis. The only drug of any value is ammonia, but this cannot be administered by the mouth when the power of swallowing is lost. In one desperate case after parturition the writer injected a diluted solution into the carotid with most striking results, the patient becoming conscious in a short time, and the paralysis disappeared, but a week afterwards another attack occurred which was rapidly fatal. In syphilitic cases iodide of sodium in 30-gr. doses should be given in combination with ammonia ; and if the syphilis is recent, mercurial inunctions should be employed. The after-treatment is the same as in cerebral hæmorrhage. The condition of the heart should receive attention, but the practice of administering large doses of digitalis with the view of stimulating the ventricular contractions is very questionable, as this may do more harm than good by causing contraction of the small vessels. The ammonia will meet all requirements by stimulating the heart and diminishing the tendency to blood-clotting, and possibly it may cause solution or disintegration of the clot. Purgatives are objectionable for the same reasons as is venesection, and, moreover, they entail movements or disturbances of the patient's position.

THROMBOSIS IN THE CEREBRAL VEINS AND SINUSES

Etiology and Pathology.—The clotting may be *primary*, owing to the condition of the blood circulating in the sluggish manner which is normal in the longitudinal sinus. The condition is met with in infants and children suffering from prolonged and exhausting diarrhœa ; but it may follow any protracted illness, and is not very rare in the feebleness resulting from old age, and sometimes it occurs in chlorosis and

anæmia. This primary form is non-infective, and inflammatory action plays no *rôle* in its production.

The common form of sinus-thrombosis is that which is *secondary* to disease of the middle ear or mastoid cells, or to disease or injury of the skull, erysipelas of the scalp, carbuncle, or ulceration in the pharynx or nose, or to tubercular meningitis. In ear-cases the lateral or petrosal sinuses are usually affected by the direct extension of the inflammatory action to the wall of the sinus, or by means of clotting in some of the veins opening into the sinus. The secondary form of sinus-thrombosis is always the result of an invasion by streptococci or staphylococci, which excite inflammatory action in the walls of the lateral or cavernous sinuses, eventuating in clotting, which tends to become suppurative.

Symptoms.—These are variable and often latent, in the primary form attacking the longitudinal sinus in children. The only signs may be marked prominence of the fontanelle, vomiting, and convulsions, passing into coma; but œdema of the scalp and distension of the nasal and scalp veins and epistaxis may be present with strabismus and rigidity of the neck and limbs. In chlorotic subjects the advent of dulness, stupor, headache, and eye-paralysis, œdema of the scalp, and the discovery of phlebitis in the superficial veins elsewhere, point to thrombosis of the longitudinal sinus, and this is strengthened by the presence of optic neuritis. The symptoms are always local, no general infection ever occurring.

In the secondary form the brain-symptoms may be entirely absent at first, or they may be those of the meningitis which accompanies it; later on the signs of septic infection or cerebral abscess may be added, and it is noteworthy that most of these cases end by the supervention of general pyæmia, with deposits in the lung. The onset may be sudden, and the presence of an old otorrhœa, with swelling of the veins and painful œdema over the mastoid, followed by rigors and other evidence of severe septicæmia, renders the diagnosis of thrombosis of the lateral sinus highly probable. Optic neuritis may be present, and the internal jugular may be felt to be indurated, hard, and tender.

When the cavernous sinus is the seat of thrombosis, the eye-signs may settle the diagnosis. The globe becomes prominent, there is œdema of the upper lid and conjunctiva, the veins around the orbit are distended, and there may be swelling over the bridge of the nose, forehead, and temples, and paralysis of the eye-muscles. The ophthalmoscope usually reveals some papillitis and hæmorrhages into the retina, or distension of the retinal veins may be observed.

Diagnosis.—The presence of a recognised cause is of very great importance in the diagnosis of a condition surrounded by much difficulty. In infants and chlorotic subjects the

primary thrombosis is simple, or non-infective. In the secondary form the infective nature of the thrombosis is characteristic, and the presence of a local cause or focus is the chief point. The diagnosis, if doubtful, is rendered certain when pulmonary or hepatic pyæmia becomes established. In some rare cases the prominent symptoms may be those of an eliminatory diarrhœa with very offensive motions and tympanitis, and a pyrexia with septic rash, which has been mistaken for typhoid fever. The most distinctive symptom in all cases of sinus-thrombosis is the external œdema, and this should be carefully watched for; its presence, with prominence of the fontanelles, readily excludes the so-called hydrocephaloid condition commonly seen in the diarrhœas of infancy. Often the prominence of the fontanelles, when these had been previously depressed, is highly characteristic in the primary cases in infants.

Prognosis.—Both primary and secondary sinus-thrombosis are generally fatal; occasionally a recovery in the so-called 'marasmic' or infantile form occurs. The great majority of the chlorotic or so-called 'autochthonous' cases end fatally in about fifteen days.

The prognosis is better in the septic or secondary cases, if early operation be resorted to. Many cases formerly regarded as hopeless are now recognised as coming within the sphere of operative surgery.

Treatment.—This in the primary cases must be directed to the diarrhœa, anæmia, or prostration caused by the source of malnutrition. The position of the patient in bed must be such as will assist the circulation in the affected sinus, and when the longitudinal is involved the head must be kept extended. The only hope from any drug-treatment lies in moderately large doses of ammonia, either in the form of carbonate or as sal-volatile, given every three or four hours with iodides.

The treatment of infective or secondary thrombosis must be the early removal of the necrosing bone or septic focus causing the disease. In mastoid disease from ear-inflammation, the antrum and cells are to be cleared out by chiselling, and the lateral sinus exposed and opened after ligature of the internal jugular vein. The sinus-contents are then to be thoroughly removed and the space packed with aseptic gauze.

CEREBRAL HÆMORRHAGE

This affection is sometimes named 'Sanguineous Apoplexy' and often loosely termed 'Apoplexy,' but this latter condition, as just seen, is common to several distinct pathological conditions.

Etiology, Pathology, and Morbid Anatomy.—Any artery in the brain may give way and become the source of a hæmorrhage. The one most frequently affected is a branch of the middle cerebral, which itself may be regarded as the continuation of the internal carotid. The branch so liable to rupture is one which comes off at right-angles to the trunk, opposite the anterior perforated spot; it passes between the lenticular nucleus and the internal capsule, to end in the former. The arteries of the brain have few anastomoses; those of the cortex are, however, more numerous than those of the deeper vessels; the branches going to the great basilar nuclei must be regarded as terminal, possessing practically no communications with the cortical vessels. These facts explain many points in the localisation of cerebral hæmorrhages. The veins are seldom the source of hæmorrhage into the brain-substance.

Prior arterial disease plays the chief *rôle* in hæmorrhage, and in the study of the causation of the affection the most important point is the consideration of the causes which are at work in determining disease of the blood-vessels, for, roughly speaking, it may be safely assumed that rupture of a healthy artery in the brain-substance never occurs. Rupture of the middle meningeal does take place when the vascular walls are healthy, but only as the result of severe mechanical injury; and the subject of meningeal hæmorrhage will be dealt with later on. Cerebral hæmorrhage is an affection belonging to the degenerative period of life; it is rare under forty, and nearly half of the cases occur between this age and fifty, and it is frequent up till old age. It is more common in men, and is often associated with chronic interstitial nephritis and hypertrophy of the left ventricle. In other words, cerebral hæmorrhage is most frequent at that period of life and under those conditions which tend to produce the degenerative changes in the blood-vessels of the body described under 'Arterio-sclerosis.'

Atheroma is the condition constantly found. The diseased vascular wall becoming weakened by the endarteritis, it ultimately gives way under the high blood-pressure and hypertrophied heart which nearly always accompany the sclerosis. In many instances the final rupture is brought about through the intervention of miliary aneurisms, which are minute dilatations of the atheromatous walls of the small vessels, and one or more of these give way under the abnormally strong force of the hypertrophied ventricle, possibly exaggerated under some violent emotion or physical exertion.

In the comparatively rare cases of cerebral hæmorrhage in young subjects, where no atheromatous vascular changes are observed, it is believed that there probably has been some previous embolic blocking, which has led to aneurismal

dilatation on the cardiac side of the emboli ; this is the explanation of the cerebral hæmorrhage so often seen in ulcerative endocarditis. Hereditary weakness of the vital rubber—a term used by Allbutt—may explain the frequency with which hæmorrhage into the cerebral substance has been noticed to prevail in some families.

In a considerable percentage of cases of cerebral hæmorrhage, in addition to the evidence of diseased vessels and the history of the causes which produce this—viz. prolonged strain, alcoholism, syphilis, &c.—there will be found concomitant interstitial nephritis and cardiac hypertrophy. The hæmorrhage, if it be large, lacerates or ploughs up the cerebral tissue by its increasing hydraulic power, and extends in the direction of least resistance until it ruptures into the ventricles, invading the lateral, third, and even the fourth chamber.

Where the bleeding is small in amount, the hæmorrhage may gradually push aside the nerve-fibres in the white substance ; and if coagulation has time to take place, the leak is stopped before much laceration occurs. The clot may gradually alter in colour and consistency and become changed into a small cyst, which finally becomes absorbed, leaving a yellow cicatrix behind. Any considerable destruction of cerebral tissue is followed by the usual zone of red, and ultimately yellow, softening, extending into the healthy tissue. In hemiplegic cases, secondary degeneration is found afterwards, when the patient survives for any length of time, extending downwards in the motor tract through the peduncle and pons, crossing over in the medulla to the pyramidal tract of the opposite side of the cord, as well as involving the direct tract on the same side. As already stated, the most frequent site of hæmorrhage is in the region supplied by Charcot's artery, a branch of the middle cerebral, a rupture of which causes the typical apoplectic seizure, accompanied by hemiplegia. The extravasation occupies the vicinity of the corpus striatum and optic thalamus, but the paralytic symptoms are not the result of the direct injury of the basal ganglia, but are caused by the pressure on or injury of the motor tract in the internal capsule. The pons suffers next in frequency as the site of cerebral hæmorrhage ; cerebellar and primary ventricular hæmorrhages are rare. Hæmorrhages in the vicinity of cerebral tumours are commonly seen.

Meningeal hæmorrhage is generally the result of rupture of the meningeal arteries by violence, in which case the extravasation will be found to be extra-dural—viz. between the membrane and the bone ; when sub-dural, as is seen in dementia and in blood-diseases like purpura and scurvy, the vessels are either diseased or the blood so altered in its physical characters that it readily leaves the vessels. Meningeal hæmorrhage is common in newly born children owing to

complications at birth, and it is the cause of the paralysis met with in these cases, owing to its pressure upon the cortical centres. It may be also an accompaniment of pachymeningitis.

Hæmorrhage into the arachnoid space sometimes occurs from rupture of diseased cerebral vessels which have become aneurismal.

Symptoms. — The symptoms of cerebral hæmorrhage resemble those already described in embolism. There are no premonitory indications as a rule; when such occur they are due to some change in the circulation produced by the atheroma, and not to the result of the hæmorrhage, unless this has been preceded by a minute extravasation. Epistaxis, however, is not a rare feature in the previous history. The onset is sudden, and there is generally more or less loss of consciousness, the condition being described as apoplexy or an apoplectic stroke. The loss of consciousness may not be complete at first, but it generally rapidly deepens into coma in the so-called 'ingravescent' cases, or it may be profound from the first. The patient lies, in the typical instances of an apoplectic seizure, in profound coma, from which it is impossible to obtain any response by shouting into his ears or attempting to arouse him by pinching a sensitive area of his skin. His reflexes are abolished, and touching the conjunctiva elicits no movement of the eyelids. The limbs are flaccid, the face is often cyanosed and the skin covered with moisture. The pulse may be strong—it is often slow and of high tension—and the carotids may be throbbing; but sometimes the pulse is soft, though full and slow. The respiration is laboured and noisy, with tracheal râles, and the cheeks are puffed out in expiration; stertor is common, and sometimes Cheyne-Stokes breathing is witnessed in fatal cases.

The condition of the pupils varies: they do not respond to light; they may be dilated, contracted, or unequal. There is sometimes noticed a characteristic position of the eyeballs and head, known as conjugate deviation, in which both globes are strongly turned to one side—looking towards the lesion in the brain (if the hæmorrhage be into the pons, the deviation is in the opposite direction to the lesion when the nucleus of the sixth nerve is damaged). The temperature is generally subnormal, but if life continues there is soon a marked rise, which in hæmorrhage into the pons, medulla, or ventricles may amount to hyperpyrexia. In some basilar cases it may be high from the first. The urine and fæces are passed involuntarily.

An examination of the muscles during the profound coma may give no evidence of paralysis, but often some movement of the arm or leg may be detected on one side, showing that the other side is paralysed; or by lifting up a limb and

allowing it suddenly to fall, a marked difference in the tone of the muscles may be observed, which show total loss of power or some rigidity on the paralysed side.

Convulsions, if present, point usually to the cortical centres or to the pons. Death may occur in rare cases within a few minutes from the onset, but it is never instantaneous; generally several hours elapse, and patients recover sometimes after being unconscious for twenty-four or forty-eight hours, or longer. In some cases there may be no loss of consciousness, but some headache or a slight dazed feeling; and patients sometimes affirm that they had no sensation of anything being wrong till they attempted to move the paralysed limb or to speak, though such patients may some minutes afterwards pass into coma. Sometimes the attack occurs during sleep, and the patient awakes to find that he has lost power over the limbs on one side of his body.

When the unconscious period is prolonged for days, symptoms of inflammatory reaction become marked, so-called 'early rigidity' of the paralysed limbs appears, large bed-sores may rapidly form, and a low type of pneumonia develops before death.

The presence of a one-sided paralysis, suspected or partially demonstrated during the comatose condition, in the majority of cases becomes obvious as the patient recovers consciousness.

Hemiplegia is the name given to this characteristic form of paralysis; it may pass off soon after the coma in mild attacks, but it generally remains more or less permanent in several of its features. It always results when the injury caused by the hæmorrhage has occurred to the internal capsule at the most common site—*i.e.* in the vicinity of the corpus striatum; but hemiplegia follows injury to any portion of the motor tract, from the motor centres in the cortex of the brain to the upper part of the pons Varolii. Certain peculiarities may, however, be noticed, depending upon the exact localisation of the damage, whether it be in (1) cortex; (2) corona radiata; (3) various points in the internal capsule; (4) crus cerebri; (5) pons; or (6) medulla. There is loss of power in the muscles of the face, arm, and leg on the side of the body opposite to the brain-lesion, when the hemiplegia is said to be *complete*. It is obvious that other conditions than cerebral hæmorrhage may produce identical symptoms—viz. thrombosis, embolism, tumour, abscess, depressed fractures, compression during birth, and meningeal hæmorrhage or inflammation. The hemiplegia is said to be *incomplete* when only a leg or arm, or the face or tongue is involved. This is the case when the cortex or white substance of the hemisphere is involved. It is also termed *monoplegia*.

The paralysis of the face affects chiefly the lower zone, the upper region being only slightly weakened, so that the

patient can close his eyes and wrinkle his forehead, though he cannot blow out his cheeks, the angle of the mouth being drawn to the sound side. The tongue is usually affected; when protruded it deviates towards the paralysed side, being pushed over by the sound genio-hyoglossus. The muscles of the trunk on the affected side are somewhat weakened, but never paralysed in the ordinary sense. This may be noticed in the intercostals when the patient attempts a deep breath, and in the abdominal muscles when he coughs or attempts to sit up, though in ordinary quiet breathing the respiratory movements may be greater on the affected side. The explanation of the escape of the trunk-muscles and the masseters, first given by Broadbent, is generally accepted—*i.e.* that the muscles which are always bilaterally associated in their actions, as the intercostals of both sides, are represented in both hemispheres (owing possibly to the existence of commissural fibres in the spinal or bulbar centres), whilst movements which are unilateral are only capable of being excited in one hemisphere—*i.e.* that of the opposite side.

This hypothesis affords an explanation of most of the features of hemiplegia. By it we can understand how the bilaterally associated movements of the upper part of the face escape obliteration; and it also explains how the legs suffer less than the arms, being much more closely associated with each other in action (as in walking) than the upper extremities are. It also affords a key to the explanation of the phenomenon of *double* hemiplegia, or pseudo-bulbar paralysis; in ordinary hemiplegia, swallowing and articulation being bilaterally associated movements, they escape, but when the hemiplegia is double, paralysis is marked as in true bulbar paralysis. In cases of right-sided hemiplegia, the hæmorrhage being into the left side of the brain, there is often *aphasia*, owing to injury of Broca's convolution; but in persons who are left-handed the aphasia may accompany hæmorrhage into the right hemisphere. Sensation is seldom as profoundly affected as motion, but complete hemi-anæsthesia is sometimes met with where the lesion affects the sensory path in the internal capsule, in the posterior third of its posterior limb; it may occur to a limited extent also in cortical lesions, and as a rule it is more complete in softening from emboli and thrombosis than in rupture of a vessel. The loss of sensation to touch or to pain may be scarcely observable, and as a rule it is most marked in the extremities of the paralysed limbs, and may be associated with hemianopia; as a rule, both these rapidly pass away. When the hæmorrhage is into the crus, medulla, or pons, there may be paralysis of a cerebral nerve on the side opposite to the hemiplegia—a condition known as *crossed* hemiplegia. Thus in lesions of the lower part of the pons the facial is

damaged on the side opposite to the hemiplegia; and in lesions of the crus the eye-muscles on the side opposite to the hemiplegia are involved. There is usually slight impairment of the muscles on the sound side in most cases of severe hemiplegia; this is generally most marked in the leg.

The paralysis of the face, arm, and leg may pass off rapidly in a few days if the damage has been very slight; but as a rule weeks elapse before marked improvement sets in, even in the most hopeful cases. The leg recovers before the arm, and the shoulder-muscles before those of the hand, and it is not unusual to see a hemiplegic patient walking with his arm helplessly hanging by his side. Recovery first begins in certain groups of muscles in the paralysed limbs; the facial muscles as a rule rapidly recover. The extensors of the foot and the knee-flexors are more deeply involved than the other leg-muscles, and they are usually more or less permanently paralysed, so that the toes cannot be raised above the ground, and progression is accomplished by a semicircular movement of the leg. Recovery is slower and less complete in the hand and fingers, which often remain permanently paralysed.

In severe cases which do not recover, certain changes are generally observed, chief amongst which is *rigidity*. Rigidity may be often observed during the coma. So-called *early rigidity* is often noticed during the first week or two; it is caused by some irritation of the pyramidal fibres or by an inflammatory action around the clot; it may pass into the *late rigidity*, which is probably the result of the descending degeneration which follows the injury to the motor tract, and it may last for years, or pass into permanent structural contracture. In the upper extremity the spastic condition of late rigidity is seen by the adduction of the shoulder, flexion of the elbow and wrist, and of the fingers into the palm; in the lower extremity the knee is straight, and the foot is in the position of equino-varus. The muscles do not show wasting for some weeks, and then generally it is very slight and is owing to inaction. Their reactions remain normal for a long period, but the deep reflexes are exaggerated. During the coma they are all abolished, but soon return after consciousness on the sound side, when they are somewhat increased generally; later on they appear in the affected limbs, where they are always much exaggerated after the end of the second week. On the affected side the knee-jerk is very excessive, and ankle-clonus is easily elicited, and the great toe, on tickling the sole, is extended (Babinski's sign), and a tap on the front of the wrist causes active flexion of the fingers. The superficial reflexes on the weak side are generally greatly diminished. With the rigidity is often associated much pain, and though the flexion may be overcome by extension it immediately recurs. The contraction at first disappears during sleep, and may be

removed by Esmarch's bandage ; later on, when the structural contracture supervenes, the joints become permanently immovable. Tremors, spasms, and irregular movements of various kinds occur chiefly in the arms in many of the cases which recover, or partially recover.

Athetosis, or hemiathetosis, is the name given to a mobile spasm which affects the arms and hands in young subjects, but which also may involve the toes. In the hand the interossei and lumbrical muscles are in a state of rhythmical activity ; the fingers are extended and widely separated, after which they are made to approach each other before being slowly flexed, as graphically described by Gowers, who likens the motion to that of the tentacles of a cuttle-fish, the wrist slowly flexing and extending. The face generally escapes. The spasms are intensified by movement and emotional activity ; when long established they continue during sleep. They are more frequently the result of embolic or thrombotic softening in children, and are rare after cerebral hæmorrhage in adults. There are in some cases symptoms of marked incoördination on voluntary movements, and the condition has been termed *post-hemiplegic chorea*.

Convulsions occurring at the onset or during the coma are comparatively rare ; they generally indicate cortical or subarachnoid hæmorrhage, but they may appear in the late stage of hemiplegia, when they are sometimes *focal*, indicating at times the site of the old lesion, the convulsion being confined to the paralysed side.

Infantile hemiplegia is the name given to a form of paralysis occurring in children at any time from birth to eight or ten years afterwards ; the great majority of cases, however, occur before the termination of the second year. It usually begins with convulsions and loss of consciousness, and the hemiplegia is generally complete.

The paralysis may pass off entirely, but generally it leaves some permanent marks behind. Thus, post-hemiplegic rigidity with athetosis is common, and the use of the limb is endered almost impossible. Marked stunted growth often esults in the affected side, and there is very often serious mental weakness and atrophy when sclerosis of a lobe or entire hemisphere follows.

Porencephalus, or loss of cerebral tissue which is converted into large cavities or cysts, is found often after death in long-standing cases, but there is never the characteristic wasting seen in the muscles in the ordinary form of infantile paralysis. Focal convulsions and epilepsy are common sequels.

In addition to the facts just mentioned, the following points are of value in the localisation of the lesion. When the hæmorrhage is *ventricular* the coma is profound and the paralysis is general ; convulsions are often present, or in their

absence twitchings and marked rigidity may be obvious. Often the invasion of the ventricle may be diagnosed when the previous symptoms indicative of the common form of internal-capsule hemiplegia suddenly deepen and become general, with coma of a profound type, as the blood first distends one and then the other lateral ventricle and passes into the third and fourth chambers. The pulse may fall to forty or fifty, and the respirations become very laboured. An extensive hæmorrhage into the central region of the *pons* causes bilateral or general paralysis, deep coma with pin-hole contraction of the pupils and hyperpyrexia which falls speedily, death occurring within fifteen minutes from the onset. The conjugate deviation of the eyes may be different from that usually seen in ordinary hemiplegia, and this is characteristic when the lesion is a small one, producing an otherwise typical hemiplegia.

In hæmorrhage into the *crus*, in addition to the crossed hemiplegia already described, when the extravasation is extensive, it passes upwards, involving the foot of the internal capsule, causing hemianæsthesia, and downwards into the pons, causing bilateral paralysis of the third nerve and of the limbs.

Medullary hæmorrhage is usually fatal, sometimes within a few minutes, the patient sinking in a profound coma without localising symptoms ; should he recover the first shock of the hæmorrhage, bulbar paralysis may be observed.

Cortical hæmorrhage is usually accompanied by convulsions, which commence in the region represented in the cortex, and the subsequent paralysis is often of the type of a monoplegia.

Meningeal hæmorrhage varies greatly in its symptoms according to the causes at work. Convulsions are common, and delirium supervenes early, but often there are premonitory symptoms, as headache and giddiness ; the paralysis is usually general in distribution, and there may be ocular palsies as well. In traumatic cases there is sometimes complete recovery from the initial shock ; this may last for many hours before a slow ingravescent stupor is succeeded by a profound fatal coma, with or without convulsions.

Diagnosis of Cerebral Hæmorrhage.—When the patient is first seen in a profound coma the difficulties are great, especially when there is no history, as in many cases found unconscious and brought to the hospital extern room. The question to be first settled should be, Is the case one of real apoplexy caused by some cerebral lesion, or is it one of uræmic or diabetic coma, or is it the result of poisoning by alcohol, opium, or chloral, or is it a case of head-injury, epilepsy, hysteria, or syncope ?

The characteristic features of cerebral apoplexy have been described on page 176, and the observer, by watching for a

few moments, may be able to determine that the apoplexy is cerebral if he sees that there is evidence of hemiplegia. The patient in his coma may move the limbs on the sound side of his body only, and the physician, by lifting up the arm of the suspected side, may find that it is absolutely powerless; compared with the opposite member, it has lost all tonus; or it may be rigid, or it may be the seat of twitchings or of a unilateral convulsion. The cheeks may puff out only on one side, and some other irregularity of the face may be noticed; upon lifting the eyelid conjugate deviation of the eyeballs may be noticed, when the diagnosis will be certain. If the coma has lasted for several hours, the reflexes may have returned to the sound side, and the plantar and cremasteric may be demonstrated on one side and not on the other.

Head-injury is then looked for if the above rapid survey fails to reveal hemiplegic symptoms, and an examination of the scalp may show a wound or a depressed fracture. It must not be forgotten that many apoplectic patients brought to hospital present contusions on the scalp or temples, caused by the fall on the stones of the footway in the street, the result of the apoplectic stroke. Rupture of the middle meningeal is often caused by violence, and the site of the wound or contusion should be considered.

Alcoholic poisoning should always be suspected in cases of difficulty; but grave mistakes are constantly made in coming to a conclusion from the odour of the patient's breath, as he may have taken alcohol upon feeling the first symptoms of his attack, or he may be suffering from cerebral hæmorrhage, induced by his being already over-stimulated by the drug. The pupils are generally dilated; and though this is a valuable point in the diagnosis from opium-poisoning, it is of little value in excluding brain-hæmorrhage, because the pupils may be found dilated in this condition also. Success in rousing the alcoholic patient need not be considered, as this is impossible when the coma is profound. There is, however, a valuable aid, generally overlooked, which is especially reliable in the class of drunken case picked up on the roadside or found comatose in bed. This consists in the marked drop in the temperature, which may amount to 5° or even 10° F. It may seem a serious matter to use the stomach-pump on a patient with cerebral hæmorrhage; but it is really a very serious matter to permit a case of severe alcoholic poisoning to die from the effect of an overdose, which might have been removed with ease; and in all cases of grave doubt the passage of the soft rubber tube into the stomach, without changing the patient's position, should be resorted to.

Opium-poisoning is recognised by the odour of the breath when laudanum is the form; the pupils are contracted to a pin's point, and the breathing is slow; of course, all the

symptoms are bilateral, and the coma, at first incomplete, becomes profound. It resembles hæmorrhage into the pons, but the temperature is below the normal, and there are no convulsions.

Chloral-poisoning closely simulates apoplexy ; the pupils are contracted, but not to the extent seen in opium-poisoning ; the pulse is weak and slowed ; the depression of the temperature is characteristic : there may be a fall of 10° F. or more.

Uræmic poisoning is a common cause of unconsciousness, though it is, perhaps, rare for the coma to be very profound. It very often follows, or alternates with, convulsions, and these are never unilateral. The high-tension pulse, hypertrophied heart, and œdema may be generally observed, and albumin is found in the urine ; but these are very often present in cerebral hæmorrhage, as they are indications of diseased vessels. Notwithstanding, also, the fact that a cerebral hæmorrhage may cause albumin to appear in the urine of a patient without kidney-disease, an examination of the urine often may settle the diagnosis. If the catheter removes only a very small amount of urine of very low s.g., the diagnosis of uræmia is almost certain to be correct. A bladder distended with a high s.g. urine, though highly albuminous, is not indicative of uræmic poisoning.

Diabetic coma has very frequently more the aspect of a complete collapse with stupor than of a profound coma ; but in cases where the onset is very sudden it may resemble apoplexy ; the breath has a peculiar sweet odour ; the urine is saccharine, and β-oxybutyric acid can be detected in it by lævo-rotation. In some cases the characteristic slow dyspnœa may be present. Even with all these characters present there should be a search made for hemiplegia, as cerebral hæmorrhage is not uncommon towards the late stages of diabetes.

Epileptic coma is less profound than apoplexy, as a rule, and soon passes off ; it resembles deep natural sleep, and the reflexes rapidly return. Transient hemiplegia and fleeting aphasia are, however, not very rare after epileptic convulsions.

General paralysis of the insane is often characterised by a series of attacks of coma resembling cerebral hæmorrhage, and the diagnosis in the absence of a history is difficult ; but soon the attack passes off, leaving the patient in his former condition, with grandiose delusions, tremors of the face, &c.

Congestion or *cerebral hyperæmia* sometimes produces a condition approaching apoplexy ; but the unconsciousness is never so profound, and it rapidly passes off without hemiplegic symptoms.

Hysterical coma is recognised by the other symptoms of this diseased condition ; consciousness is not really lost, the pupils readily respond to light, stertor is absent, and the symptoms rapidly yield to external stimuli

Syncope is easily eliminated, the cardiac failure being the chief feature, as evidenced by the absence of pulse and the feebleness of the heart-sounds; the sphincters do not give way, and the reflexes are rarely lost.

Assuming the diagnosis of cerebral apoplexy with hemiplegia to be demonstrated, the question then comes to be whether the cause is one of hæmorrhage, embolism, or thrombosis; complete loss of consciousness may characterise each, and the history of the case is generally necessary before the lesion can be determined.

The following points may be repeated: In *embolism* the coma will be less profound; as a rule, the patient will probably be under forty; there will be probably heart-disease or arterial plugging elsewhere present; the onset is rapid, and the symptoms may pass off quickly. Should the question of diagnosis only arise during the hemiplegic state, the fact of a sudden seizure without unconsciousness is in favour of embolism.

In *thrombosis* the differential diagnosis is most important, as it seriously influences treatment. It may be very difficult, as evidences of diseased vessels are to be found in both cases, and age affords little help. The history of the onset is of vital importance. It lacks the suddenness of hæmorrhage and of embolism; there are premonitory headache, vertigo, and tingling in the limbs on one side; consciousness, if lost, is lost slowly; in syphilitic cases it is often retained. The state of the pulse is important; the strong heaving impulse of hæmorrhage is absent, and the pulse is weak and of low tension. Local convulsions at the beginning of the attack are more likely to occur in thrombosis, and general convulsions in hæmorrhage. The temperature, unless the thrombosis be very extensive or basilar, is not influenced, as it is in hæmorrhage, in which latter condition there is usually a sudden moderate fall.

Abscesses, *tumours*, and *aneurisms* may produce hemiplegic symptoms with more or less interference with consciousness; these are described under their respective headings. The rupture of a large cerebral aneurism may resemble closely in its symptoms a large hæmorrhage into the ventricles, and the diagnosis can only be suspected by a consideration of the former symptoms.

Prognosis.—As regards recovery from the coma, the prognosis is always very serious; it is grave in proportion to its depth and duration, and if the loss of power is evident on both sides death is probable, and practically certain if Cheyne-Stokes respiration and profound coma are present at the end of twenty-four hours. A sudden and rapid oscillation of temperature is of evil omen; so also is a bounding pulse and flushed face with much stertor. In meningeal hæmorrhage from injury, surgical treatment is often successful.

The hemiplegia which remains, in the majority of cases becomes permanent. If it is to get well, as a rule, it does so quickly, and if there be not marked improvement in ten or twelve weeks it will probably be permanent, though the patient may be able to walk about ; but the upper extremity will be of very little use to him, and when contracture has developed the prospect of further improvement is hopeless ; and even as soon as late rigidity shows itself there is little hope of any marked improvement. Some mental weakness often remains.

Treatment, for the comatose stage, consists in absolute rest, with ice to the head and counter-irritation to the nucha, a brisk purge with croton oil, and when the pulse is bounding and tension high a smart blood-letting where the diagnosis positively excludes thrombosis. For the hemiplegia little can be done ; nothing should be attempted for three or four weeks, and then only in the absence of all secondary inflammatory action. Massage, faradic and the continuous currents and passive movements may be tried cautiously.

CEREBRAL HYPERÆMIA AND ANÆMIA

There are strong reasons for doubting the existence of these conditions as clinical entities, and much that has been written and attributed to cerebral congestion is believed to be the result of a fleeting alteration in the cerebral vessels, caused by embolism, slight thrombosis, or possibly minute hæmorrhages. If a real hyperæmia of the brain ever exists independently of some obstruction to the flow in the veins from pressure or heart-disease, it is likely to result from the action of some morbid poison which acts as a local vaso-dilator ; but even this is purely problematical. Nevertheless, there is still a widespread belief that the symptoms of headache, mild fever, flushing of the face, and throbbing of vessels, with some confusion of thought and transient unconsciousness, which occur in full-blooded plethoric people past middle life, are caused by hyperæmia of the brain ; and the belief is strengthened by the experience that these symptoms are speedily relieved by smart purgation or epistaxis, as they were formerly frequently treated by blood-letting. Similar symptoms seen in over-active and over-stimulated young brainy subjects are probably vaso-motor disturbances akin to migraine.

The opposite condition, of cerebral anæmia, certainly must exist in cases of profound general anæmia, from whatever cause produced ; and here it is not the quantity, but the quality, of the blood circulating through the brain which is most likely at fault, especially as in such conditions as pernicious anæmia the total bulk of the blood in the body may be increased.

This latter consideration would seem rather to tell against the theory of cerebral congestion as a special affection resulting from overfilling of the cerebral vessels.

The *hydrocephaloid* condition in children which supervenes upon an exhaustive diarrhœa, in which there are seen contracted pupils, depressed fontanelles, marked pinching of the face, stupor, and coma, is attributed to a cerebral anæmia. Such a condition will probably be demonstrated to be the result of ptomaine or bacterial poisoning.

Symptoms of a true anæmia of the brain are observed in the coma following the sudden occlusion of a large artery, and they are demonstrated in cardiac syncope. They are constantly observed after extensive bleedings and during the late stages of pernicious anæmia. If the anæmia be profound, as in syncope, consciousness is lost; if of lesser degree, there are drowsiness, vertigo, flashes of light, noises, or singing in the ears, contracted pupils, pallor, with cold moisture on the skin, and even convulsions in which the pupils dilate. When the condition is very chronic, the symptoms are those of so-called 'irritable weakness,' in which, with some of the preceding features, there are mental dulness or inability to think, mild delirium, dimness of vision, and hallucinations; all of which symptoms may only appear upon assuming an upright posture, and they are relieved or prevented by a horizontal position.

Treatment.—For the immediate relief of the symptoms of cerebral anæmia the patient should be at once placed in the horizontal position, the cause of the condition being afterwards treated upon the general principles indicated in anæmia, syncope, &c. Plethoric cases should be treated by elevation of the head, the ice-cap, sinapisms to the neck and feet, a severe purge or blood-letting.

CEREBRAL INFLAMMATION

As implied in its synonym—Acute Encephalitis—this is an acute inflammation of the brain-tissue.

Etiology and Pathology.—Acute encephalitis of the non-suppurative type is seen as a local affection in the secondary changes of red softening visible in the zone of cerebral tissue surrounding necrotic softening, hæmorrhages, abscesses, tumours, and other forms of cerebral injuries, and in a more diffuse type as secondary to meningitis.

Following general toxic conditions, as those prevailing in the infectious fevers, especially in *influenza*, and as a result of chronic alcoholism or syphilis, a diffuse form of cerebritis presenting all the characters of the above-mentioned red softening is recognised as a not very infrequent affection.

The grey matter is most seriously involved, sometimes in the cortex and sometimes in the basilar ganglia; but the white matter generally also suffers, though to a much less extent. There is congestion of the small vessels; these are surrounded by blood-cells and leucocytes, and the nerve-cells and fibres are found in all stages of disintegration in the swollen reddish patches of the diseased tissue. The condition is regarded by Strümpell as identical with that producing *poliomyelitis anterior acuta* in the cord in infantile paralysis, though the affection in the brain is not so definitely limited to the grey elements; both the cerebral and the spinal forms have been demonstrated in the same patient. The condition described by Wernicke as the *superior*, involving the floor of the fourth ventricle and the aqueduct of Sylvius, and named *polio-encephalitis acuta hæmorrhagica*, and an *inferior* variety involving the medulla, may be taken as varieties of acute encephalitis, differing only in their sites and caused by the same morbific agents, though alcohol is believed to be the most frequently active cause in the superior variety. Sometimes the condition extends in the inferior variety downwards to the cord, and all three varieties may exist together. Still another variety is described as Strümpell's *acute primary hæmorrhagic encephalitis*, which chiefly involves the cerebral hemispheres affecting the white matter, though to a less extent than the grey. It is most frequently observed as a post-influenzal process in girls. All varieties are more common in youth, except those produced by alcohol, and a chronic, more diffuse form attributed to syphilis.

Symptoms.—These vary greatly, and the diagnosis is one of much difficulty, unless the cause be obvious, as in cases following head-injury and influenza. In the *superior* acute hæmorrhagic forms in children the onset is very sudden. Head-ache, fever, vomiting, giddiness, convulsions, retraction of the head, restlessness, excitement, and delirium are soon followed by paralytic symptoms involving the eye-muscles (double ophthalmoplegia); there may be monoplegia, hemiplegia, optic neuritis, or aphasia, and somnolence appears, rapidly passing into coma with a rapid pulse and Cheyne-Stokes respiration, death resulting inside one or two weeks from the onset. In the alcoholic cases the above symptoms may come on suddenly in patients addicted to chronic excesses, the temperature being subnormal from the onset; though the ocular paralysis (ophthalmoplegia) is well-marked the sphincter of the iris and the upper lid may remain normal; nystagmus is present, and great restlessness, with cerebellar gait, preceding coma. In the *inferior* form the general initial symptoms are of the same type, but the paralysis is of the glosso-labio-pharyngeal distribution, and may be also accompanied by the various forms of spinal palsy.

Diagnosis.—The chief difficulty is in excluding meningitis, and, as pointed out by Fleming, this may not be possible in the diffuse form caused by syphilis till the acute symptoms pass off, when symptoms of mental impairment are much more likely to remain in encephalitic cases than in meningeal. The chief point in the diagnosis of acute encephalitis, as already mentioned, is the sudden onset, with ocular paralysis following a definite cause. In the *inferior* variety the sudden onset of bulbar paralysis following influenza locates the affection. Sinus-thrombosis is excluded by the absence of local infection and of œdema.

Prognosis.—This is always serious. The disease is generally fatal in the acute hæmorrhagic forms. Syphilitic cases may be controlled when their progress is sufficiently slow to admit of treatment by iodides.

Treatment.—This will consist in the exhibition of remedies indicated for the relief of the prominent symptoms; thus severe headache calls for antipyrin and the ice-cap. As a rule, the agents suitable in most cases of meningitis may be employed for the relief of delirium, insomnia, and convulsions.

CEREBRAL SCLEROSIS

As implied in its name, this is an altered condition of the brain-substance in which its consistence is considerably augmented by the great increase in the neuroglia and proliferation of the connective-tissue elements, with a corresponding atrophy or degeneration of the nerve-fibres and nerve-cells. The condition may be confined to the cerebrum, or may extend downwards along the motor path to the spinal cord, or it may be confined to the latter region entirely or may involve the nerve-roots and their cords.

The condition, with the exception of that variety of it which constitutes Disseminated or Multiple Cerebro-spinal Sclerosis, is mainly of pathological interest. The disease just mentioned, however, is a well-marked and by no means infrequently met with clinical entity of considerable importance, and will be described under its own name. It must, however, always be remembered that in rare cases this disease may be confined to the cerebral tissue, though almost invariably the sclerotic areas are disseminated throughout the grey and white matter both of the brain and spine.

Cerebral sclerosis may be congenital or hereditary, arising in a developmental error, in which case the chief or only change is an increase in the embryonal neuroglia-tissue of the white matter. This form is met with as a *diffused* sclerosis in idiots or imbeciles and may involve one entire cerebral hemisphere, or more frequently a considerable portion of it or a single

lobe. It may betray its presence early by the arrest of the mental development, in which case a typical state of idiocy results, often accompanied by hemiplegia and epileptic seizures, with various deformities caused by arrest of growth in the limbs. When the sclerotic change sets in at a later stage in life, the mental development previously attained is not only arrested, but usually retrogrades into complete imbecility.

Sclerosis of a *primary degenerative* type is believed to occur in the senile period of life, when the nerve cells or fibres in the convolutions decay, and their degeneration is followed by hyperplasia of the connective-tissue elements. A similar condition of affairs is observable in syphilis and other forms of toxæmia, though here the sclerotic change is more likely to attack the susceptible systemic paths in the cord.

Sclerosis of a *secondary degenerative* type is much more common, and is constantly seen in those pathological conditions where from any cause (disease, hæmorrhage, pressure, &c.) a nerve-fibre is cut off from its trophic ganglion-cell.

Sclerosis of a purely *inflammatory* nature and chronic type is part of the morbid change found constantly in association with the irritation produced around cerebral hæmorrhages, chronic abscesses, and tumours growing in the cerebral substance. The irritation may originate an inflammatory action in the neuroglia itself or in its connective-tissue elements, or in the perivascular sheaths of the small blood-vessels. In any case the results are the same; the neurons perish by the pressure which the new dense growth exerts upon the nerve cells and fibres.

The sclerotic patches may assume various shapes and dimensions, from the miliary and small nodular or tuberous growths which are often found in the convolutions or deeper in the brain of subjects who presented no symptoms of cerebral disease during life, to the huge diffuse sclerosis which may involve an entire lobe or hemisphere in the brain of the idiot or imbecile. The hard and toughened nodules or plaques are recognised by their greyish-white colour, when the early stage of translucent greyish-red swelling has given way to shrinking and condensation from the contraction of the new fibroid proliferation. Under the microscope the degeneration of the medullary sheaths is visible, naked axis-cylinders remaining till they finally disappear under the destructive pressure caused by the thickened neuroglia and connective tissue fibres which are everywhere evident in the section.

CEREBRAL SOFTENING.

(See p. 168, under CEREBRAL EMBOLISM.)

CEREBRAL SYPHILIS.

(See under MENINGITIS, CHRONIC.)

CEREBRAL TUMOURS

Etiology, Pathology, and Morbid Anatomy.—Any part of the brain or its membranes may be the seat of almost any type of either primary or secondary new-growth. As little is known of the causation of tumour-growth here as in any other portion of the body. Heredity and trauma are believed to account for a small proportion of cerebral tumours. They are more frequently met with in males, and may occur at any age after the first half-year of life, though certain tumours are more common at particular periods of life; thus, tubercular growths occur more frequently in children and syphilitic tumours after or about middle life. Sarcoma appears most frequently between twenty and forty, and the same is true of glioma, while carcinoma occurs at a later age. Exclusive of tubercle, speaking generally, cerebral tumours are rare in the very young and in old age.

As regards the frequency of the different forms, as determined by *post-mortems*, tubercle heads the list, being about as common as all the other forms together. Sarcoma and glioma come next. Many of the syphilitic cases, being curable, do not figure in *post-mortem* returns.

Tubercular growths occur as spherical masses in size from that of a pea to three or more inches in diameter, consisting of caseous yellow material, formed from the degeneration of miliary granulations. Spots of softening or calcification may be observed in their interior; the outer layer of the growth is translucent and greyish in colour, and it lies in a bed of softened cerebral tissue, from which it is easily detached. They are most frequently found in the cerebellum; though usually solitary, they are sometimes multiple, and in many cases the lungs or lymphatic glands are also the seat of tubercle. They may remain quiescent for long periods. In tubercular meningitis small nodules of tubercle are scattered over the pia mater and along the vessels.

Sarcoma may be primary, but is often secondary. Various forms of sarcomata (fibro-, myxo-, angio-, as well as the cystic and alveolar types) are met with in the brain or cranium springing from the cerebral tissue, membranes, or bone. They may be very firm or very soft in consistence; they possess the general character of not infiltrating the cerebral substance, but in their growth push it aside, causing a zone of softening from which they are easily detached. They are often single,

and sometimes make their way when they spring from the dura towards the surface so as to be felt under the scalp, in which situation they are very large.

Glioma is a form of growth allied to sarcoma, springing from the cerebral tissue. It is generally solitary ; it grows rapidly and infiltrates the brain-substance, from which it cannot be detached. In appearance and consistence it usually resembles the grey matter, from which it is never encapsuled, and on microscopic examination the resemblance continues, its delicate cells and fibres representing elements like those found in the neuroglia. Like the true sarcomata, the gliomata vary much in consistence and structure, and an intermediate form known as a *glio-sarcoma* is not rare. The gliomata are very liable to hæmorrhages, especially the softer forms, as the myxo-glioma. They are often met with as large as a small plum.

Syphiloma—gummatous tumours in the brain—seldom appear till seven years after the original attack of syphilis. They resemble the tubercular in colour and consistence, but are irregular or nodular in outline, and often of about the size of the kernel of a walnut. They do not infiltrate ; they are found chiefly on the surface of the brain springing from the vessels of the pia and matted with the dura mater. Occasionally a gumma has been found in the interior of one of the ventricles. Their favourite site is the convexity of the cerebral hemisphere and the pons. Unlike the tuberculata, they are rare in the cerebellum and corpus striatum, and may sometimes be found connected with the trunk of a cerebral nerve.

Carcinoma is comparatively rare. It may be single or multiple, primary or secondary, and may spring from any place inside the cranium ; the encephaloid type is the most common.

Psammoma is a small tumour of little clinical significance, consisting of brain-sand held together by fibrous tissue, found in the pineal gland and choroid plexus. Myxomata, Cholesteatomata, Lipomata, Melanomata, Fibromata, Osteomata, and other rare forms, are occasionally met with.

Parasitic cysts are not very rare. They contain hydatids or cysticerci, and are commonly multiple. *Simple cysts* in various parts of the brain are occasionally met with, probably as remnants of degenerated tumours or of encapsuled hæmorrhages.

The pathological changes produced by tumours in the brain-substance are of importance, as they afford a clue to the clinical symptoms. In the immediate neighbourhood of the growth there is more or less destruction of the cerebral tissue the result of local pressure, and in the infiltrating glioma the result of direct invasive destruction of the cells

and fibres. An area of red softening generally surrounds the growth, this is the result of inflammatory or irritative pressure, and if the tumour be near the surface it may set up a local meningitis which may become general. The pressure acts also in another way owing to the rigidity of the cranial wall: the general intracranial pressure is raised, and parts at a distance from the growth are compressed even when it is situated under the tentorium, in which case additional general pressure is exercised by the dropsical condition of the ventricles, the result of interference with the veins of Galen. The convolutions become flattened, and the cranial bones may be thinned out so as to yield to the pressure of the finger; this may occur locally when a large tumour grows from the surface of the brain or its membranes, but it also may be general in the hydrocephalus caused by ventricular dropsy.

Symptoms.—The symptoms caused by cerebral tumours naturally divide themselves into two groups—the *general* symptoms, produced mainly by the increased pressure, regardless of the exact site and nature of the growth; and the *local* or so-called focal symptoms, which depend upon the site of the tumour. Occasionally a tumour of considerable dimensions has been found after death in a patient who had never shown any cerebral symptoms or other evidence of its presence. The general symptoms are *headache, vomiting, optic neuritis, giddiness, general convulsions,* and *mental disturbance.*

Headache is present in the great majority of cases, and it varies much in its character: as a rule it is intense, and often it is agonising. It may be continuous, but generally it comes on in severe paroxysms during the intervals between which more or less pain is also commonly experienced, but sometimes the patient may experience complete relief in the intervals. It may be localised, corresponding to the site of the growth, when this is superficial, in which case local tenderness on percussion may be present also; but deep-seated tumours may cause pain in distant parts. Though, roughly speaking, frontal growths cause headache in the forehead, and cerebellar growths pain in the occipital region, not infrequently, a cerebellar tumour causes headache located in the opposite frontal region, and *vice versa.* The pain is probably the result of pressure, since it is often relieved by trephining, and it is generally aggravated by coughing, sneezing, or deep inspirations.

Vomiting and *giddiness* are frequently associated with the headache, and may be present with tumours in almost any situation. The vomiting is paroxysmal, and in some instances very severe; it is not usually attended with much loss of appetite and occurs with a clean tongue. Sometimes there is an

enduring nausea ; whilst at other times the food is rejected almost without any feeling of sickness. It is almost always present in *sub-tentorial* tumours, being especially constant when the middle lobe of the cerebellum and the medulla are involved. Giddiness, or vertigo, is not so frequently present as vomiting. It may be produced by paralysis of the eye-muscles or irritation of the auditory nerve ; it is especially marked when a tumour affects the middle cerebellar peduncle, pons, medulla, or corpora quadrigemina.

Convulsions of a general type are not uncommon, and they are identical with epilepsy, even to the extent of presenting the characters of petit-mal, and may occur in tumours affecting any region of the brain. Different, however, are the local convulsions known as Jacksonian epilepsy, which are to be regarded as belonging to the focal or localising symptoms.

Mental disturbances, from mere hebetude and loss of memory to delusions, hallucinations, and dementia, may be present ; they may occur with a tumour in any region, but are believed to be more frequent in frontal cases. Slowness of speech may be often present without any pressure upon the speech-centre regions.

Optic neuritis is mentioned last, though really the most important, because the previous symptoms are apparent or obtainable from the patient. Of the presence of optic neuritis he is wholly unaware, and an ophthalmoscopic examination should always be made and repeated at intervals in every case where the presence of a cerebral tumour is suspected. The optic nerve at its intra-ocular termination in the papilla or disc is found inflamed in more than four out of every five cases ; in the great bulk of all cases the neuritis is double, though it may be more marked in one eye than in the other. Sight may be normal with very marked inflammation of both discs, but when consecutive atrophy occurs, failure of vision becomes more or less marked ; but the atrophy is a slow process, and does not usually appear for many months after the onset of the neuritis or papillitis. Optic neuritis is more often absent in tumours affecting the upper part of the cerebrum or its membranes ; it is not the result of general pressure alone, but is probably also caused by irritation affecting the optic tract and nerves. Hence, though its presence is of little localising value, it is more intense and rapid in its destructive course in tumours of the cerebellum, and in the region of and involving the optic tracts. It may become stationary or very slow in its progress in chronic cases ; but in quickly growing tumours the optic neuritis is generally very rapid in its course, and atrophy is almost certain to follow. Its severity is no indication of the size or nature of the growth, as Gowers reports its absence in a case where a sarcoma as large as the fist grew from the dura, compressing the middle

of one hemisphere. It may supervene suddenly and with great intensity during the course of the most chronic growth. There may be blindness without optic neuritis in tumours involving the optic tract.

The *local* or *focal* symptoms by which the site of the cerebral growth is localised are numerous. The more important are paralysis, localised convulsions or spasms, disturbances of sensibility, and interference with the gait.

Paralysis may be of the ordinary hemiplegic type when the tumour interferes with the motor tract in the cortex ; it is rarely complete, the leg often being less affected, and it is often associated with convulsions commencing in the weakened muscles. The hemiplegic symptoms, whilst resembling those following hæmorrhage, always differ in their slow and very gradual onset. When the growth involves the lower part of the motor tract some of the cranial nerves suffer, causing crossed paralysis on the opposite side to the hemiplegia ; this is seen when the crus, pons, or medulla is involved. When the growth involving the pons or medulla is sufficiently large bilateral paralysis results, causing paraplegia and paralysis of cerebral nerves.

Optic nerve injury has been already referred to amongst the general symptoms as causing optic neuritis ; *olfactory* failure is rare. The *third* nerve suffers in tumours involving the crus, causing crossed paralysis, and the *fourth* when the region of the corpora quadrigemina is pressed upon. The *fifth* is injured in its motor and sensory portions in growths about or in the pons. The *sixth* nerve has less localising value. When affected, owing to its long course, it is generally involved in sub-tentorial tumours ; when the external rectus on one side and the internal on the other are affected the injury is in the pons, and the eyes cannot be turned towards the lesion in the brain ; when all the nerves supplying the muscles of one eye are paralysed, the growth must be in the region of the sella turcica, and the sight is also liable to be lost on the same side. The *seventh*, or facial, is only completely paralysed in tumours of the pons, when the sixth generally suffers with it.

The *spinal accessory* is paralysed by growths in the medulla, often suffering with the tongue-nerve ; but when the neck-muscles are affected the pressure must be outside the medulla, and one vocal cord may be affected.

The *auditory* nerve is involved in growths in the pons and base of the brain ; the phenomenon of word-deafness occurs when the temporal convolution is involved, and is described under ' Aphasia.'

The *hypoglossal* generally suffers with the spinal accessory in medullary growths and in lesions of the motor tract anywhere between the ascending frontal convolution and the

medulla. It is paralysed by growths in the posterior fossa of the base, about the front of the occipital bone upon the side opposite to the hemiplegia.

Focal convulsions.—Already general convulsions and also petit-mal have been referred to as amongst the symptoms caused by a cerebral tumour situated in almost any region. *Focal* convulsions are of great localising value ; they constitute Jacksonian epilepsy. The convulsion begins with twitchings, which rapidly spread to all the muscles in one limb, and the convulsion may be confined to this limb, the discharge evidently being limited in the motor area to the centre belonging to the convulsed member, whether this be the side of the face, the arm, or the leg, in which case consciousness is retained. In many instances the local convulsion spreads to the entire side, and it may cross over and involve all the limbs, ending in a general convulsion with loss of consciousness. In either case its local commencement, coupled with other symptoms of brain-disease, indicates the site of a cerebral tumour producing a cortical lesion of an irritative kind. Often a temporary paralysis follows the local convulsion, or, if the convulsion has become general, the part in which the convulsion started will often be found paralysed after consciousness is restored. Tonic spasm, or contracture of the neck-muscles causing retraction of the head, occurs in some sub-tentorial tumours.

Sensory disturbances occur. Thus hemianæsthesia may be produced by a growth affecting the posterior part of the internal capsule, or the starting-point in a convulsion may be some localising sensory disturbance.

Aphasia is comparatively rare in tumour, but both the motor and sensory forms may be present to a slight extent when the growth involves the speech-centres.

The course or duration of cerebral tumours varies greatly ; the quick-growing sarcomata may cause death inside six months, but many of the fibrous forms may exist for ten or fifteen years. Death results from the growth becoming suddenly the seat of a hæmorrhage, or from meningitis, or from pressure on the respiratory centre (when the termination may be sudden), or from coma induced by gradual pressure, or from exhaustion, the result of the intense headache, vomiting, &c.

Diagnosis.—The most important single symptom is optic neuritis ; but even when vomiting and headache are present along with it, the diagnosis is by no means certain, as these symptoms may be all present in meningitis, abscess, and inflammation of the brain ; in the majority of cases the presence of the fever and local causes, and other symptoms of these affections, will settle the diagnosis. Thus the presence of ear-disease, for example, will point strongly to abscess, and in meningitis the optic neuritis will be slight and never found

in the advanced state often seen in the first examination made in tumour cases, and the onset of the headache will be sudden. Optic neuritis may be present with headache in at least three conditions in which there is no brain-disease: (1) Anæmia; (2) Renal disease; (3) Lead-poisoning. In the first condition it may be sudden and rapid, and it may as rapidly disappear; but in any case the other symptoms of these three conditions are so characteristic that a mistake in diagnosis is unlikely, unless the rare coincidence of a tumour in the same case exists at the same time and may therefore be overlooked; this can only happen in the absence of focalising symptoms.

Epilepsy, *Hysteria*, *Menière's vertigo*, *General paralysis of the insane*, and *Disseminated sclerosis* can as a rule be easily excluded by the characteristic symptoms peculiar to each, when considered along with the absence of focalising symptoms.

The diagnosis of the seat of the growth is often very difficult; the chief localising symptoms have been already mentioned. The question is of vital importance, since only after localisation has been accomplished can the question of operative interference be contemplated. This, as a rule, is most satisfactorily carried out in *cortical* tumours, and fortunately the symptoms are sometimes well marked when the growth involves the cortex in the region of the fissure of Rolando. When the tumour destroys the centre for the arm, or lips, or face, or tongue, or the leg, the resulting limited paralysis affords strong evidence of its position; in the same way, should the centres be irritated and not destroyed by the growth, instead of permanent paralysis the characteristic convulsions may be present—viz. a convulsion confined to the part whose motor centre is irritated, or a convulsion beginning there and afterwards becoming universal. Subjective sensations in the parts belonging to these centres often precede a general convulsion, and such are valuable focalising aids, especially in cortical cases.

Tumours of the *crus* are recognised, as already stated, by hemiplegia on the side opposite to the tumour, and paralysis of the third nerve on the same side as the tumour. In growths in the *pons*, the hemiplegia is generally associated with paralysis of the facial on the side opposite the hemiplegia; the fifth and sixth nerves may be paralysed along with the facial. *Medullary* growths, in addition to hemiplegic symptoms or bilateral paralysis of limbs, will show bulbar symptoms owing to paralysis of the spinal accessory, glosso-pharyngeal, vagus, or ninth nerve, and the vocal cords may be affected.

Cerebellar tumours may give rise to no symptoms when located in the hemispheres, unless they involve the medulla by contiguous pressure, but a growth in the middle lobe produces the symptoms mentioned under cerebellar hæmorrhage, the most characteristic being the drunken or staggering

gait and so-called cerebellar ataxia with retraction of the head, nystagmus, and vertigo. When the middle peduncle is involved rotatory movements are generally present.

The presence of a tumour in the cranium, and its probable site, having been decided upon, an attempt should be made when possible to determine its nature. Syphilis and tubercle are practically the only growths about which any degree of probability in the diagnosis can be entertained. Syphilitic tumours are likely to be superficial and are rare in the cerebellum; they occur in subjects five to ten years after infection, and in congenital cases in early life. Jacksonian epilepsy is common, and when the history is absent other tertiary signs may be obtained in the bones; its growth is slow, and, more important than all these, it is influenced by energetic iodide treatment. In tubercular growths the presence of phthisis, bone or visceral tuberculosis, the early age of the patient, the cerebellar site, and the liability to meningitis, are important points; a family history of phthisis in a young subject in whom symptoms of a rapid growth occur, which afterwards tends to remain stationary for a time, is in favour of tubercular tumour. Gliomata may be suspected when hæmorrhagic symptoms supervene. A carcinoma may be suspected when a primary growth is discovered in some other part of the body; and this is true also of hydatid and other parasitic tumours and sometimes of sarcoma.

Prognosis.—This will depend upon the site, nature, and size of the growth; it is, however, always serious. Two considerations modify the gravity of the case: if the tumour be syphilitic there is a very fair hope of its yielding to iodides, and if situated in a cortical position suitable for surgical treatment complete removal may be accomplished. But treatment in those favourable cases should not be long delayed, as sudden death may supervene in any case of intracranial growth. Measures directed to the tubercular condition sometimes, but rarely, may be successful. With these exceptions, which probably only influence a small percentage of the cases of cerebral tumour, the result is always a fatal issue. Intense double optic neuritis, coming on suddenly after headache and other symptoms have been present for some time, indicates a rapidly fatal course; whilst the gradual subsidence of a neuritis in a chronic case renders the outlook more hopeful as regards prolonged duration.

Treatment.—The relief of headache and vomiting is to be accomplished by the exhibition of drugs conducted upon general principles. The ice-cap to the skull, with sinapisms to the nucha and large doses of bromides combined with antipyrin, antifebrin, phenacetin, caffeine, &c., are useful.

Iodides in large doses often afford some relief to the headache of all kinds of cerebral growths, whilst the after-disturbances

produced by morphia often prohibit the use of this drug. Leeching may be resorted to when other measures fail. Convulsions should be met by large doses of bromides combined with chloral.

In syphilitic cases iodides should be commenced without delay and pushed to the extent of 60 grs. thrice daily; mercurial inunctions are indicated in those rare cases where the symptoms come on in the early tertiary stage; but in all cases a mild mercurial should be given for short periods during the prolonged constant course of iodide treatment.

Trephining has been resorted to with very satisfactory results in rapidly growing deeply seated tumours beyond the hope of removal, with the view of affording relief from the destructive influences of the general pressure; by its means the headache, vomiting, and optic neuritis have been arrested, and the sight preserved for long periods.

When the situation of the tumour, as determined by the focalising symptoms, has become probable, an attempt should be made to enucleate the growth after the removal of a portion of the cranial table by the trephine and saw, under the most rigid aseptic precautions. The operation has been successful in the hands of Macewen, Horsley, and others; and tumours have been satisfactorily removed not only from the motor cortex, but even from the cerebellar hemispheres

CEREBRO-SPINAL MENINGITIS.

(See under MENINGITIS.)

CHELOID.

(See under KELOID.)

CHILBLAIN

This, also called Erythema pernio, is a variety of erythema produced by exposure to cold in young subjects with feeble circulation, which generally affects the hands and feet, the margin of the ears, and sometimes the extremity of the nose.

The first effect of cold is to produce a local anæmia from contraction of the capillaries. After the removal of the cause the toneless vessels dilate, and the reaction which occurs remains, the engorged capillaries and small veins becoming distended, producing a local passive congestion which leads to œdema and lividity of the affected parts. There are considerable smarting, itching, and painful sensations in the swollen region, and a tendency for the deficiently nourished skin to ulcerate spontaneously or on the slightest injury; when ulceration

occurs the affection is termed ' broken ' chilblain. Chilblain is allied to frost-bite, but gangrene is very rare, and in both cases there is an aggravation of the symptoms upon sudden exposure of the benumbed part to heat; indeed many persons can induce chilblain by a sudden exposure of a chilled part before a brisk fire. The fingers, especially the little one, and the ulnar side of the hand are most commonly affected. Upon the advent of warm weather the erythema passes off, but the ulcers are very slow to heal in most cases.

Treatment.—The condition can generally be prevented by warm coverings for the hands and feet; the use of tight thin gloves and tightly fitting light boots is to be condemned. Active exercises whilst the ' chilblainy ' subject is out of doors should be recommended, and he should be warned not to rush to hot water or hot fires upon coming in after exposure to cold. Friction of the part, gently and perseveringly applied, restores the circulation better than heat. As a preventive, and as a curative agent in the early stage, nothing is better than rubbing the parts with strong alcoholic solution of camphor. When ulcers form, a mild stimulating ointment, as unguentum resinæ, is indicated. The value of calcium chloride internally has been recently demonstrated in a number of cases.

CHLOASMA

This is the name given to the brown pigmented patches which appear in various regions of the skin during pregnancy, especially over the forehead close to the hairy scalp, extending to each cheek; the nipples and central line of the abdomen and axillæ are also generally involved. Similar pigmentation takes place occasionally in sterile women the subjects of uterine disease. The discoloration is identical with that which invades spots of the skin which have been the seat of injuries or blisters, as in the pigmentation seen in vagabonds' disease from scratching, and in the site of old scars or eruptions, or in parts subjected to the pressure of garters, trusses, and the like. The pigment is situated in the deeper portion of the epidermic layer of the skin, and, though it may remain permanently, it generally fades or disappears upon the removal of the cause. Thus it fades rapidly after delivery, but will return with each pregnancy. A strong corrosive-sublimate lotion (1 in 250), or staining the spots with the strong permanganate-of-potash liquor and washing them afterwards with oxalic-acid solution or a strong alcoholic solution of salicylic acid, will remove the affected layer.

CHLOROSIS

This, also known as Green Sickness, is an anæmia occurring in girls from some unknown defect in blood-formation, characterised by a diminution of the hæmoglobin out of proportion to the decrease of the red corpuscles, and easily curable by iron-treatment.

Etiology.—The first onset occurs between fourteen and twenty years of age, but as relapses are common the disease may be met with after thirty, though the tendency diminishes with each year.

The amenorrhœa so very frequently associated with it is the result and not the cause of the disease. Constipation, with ptomaine intoxication, tight corsets, gastric displacement, emotional excitement, aortic malformation, and other fanciful etiological factors, are but coincidents in some mysterious deficiency of blood-formation which is confined to the period during which the female sexual organs are undergoing development.

Symptoms. — There are obvious symptoms of severe anæmia, as palpitation and breathlessness upon slight exertion, languor, and a tendency to syncope. The skin and mucous membranes are blanched, the conjunctiva being pearly white or bluish white over the sclerotic, and when the condition becomes established a green shade accompanies the pallor of the skin. The bloodlessness may be masked by temporary flushing of the face, but the anæmic condition of the visible mucous membranes is always obvious. There is no diminution of the subcutaneous fat, and there is generally some slight œdema of the ankles. The menstrual secretion is scanty, or entirely absent, but rarely it may be excessive, and there is often leucorrhœal discharge, with backache. Constipation is the rule; headache, tinnitus, and temporary failure in sight and hearing are common.

Gastric derangements, indicative of dilatation and sometimes of ulcer, are present, and there is generally a marked capriciousness of appetite shown in the desire for raw cereals, acids, chalk, &c. Hyperacidity is usually complained of ; the tongue is generally clean and always pale. The pulse is frequent, weak, and compressible, and there is often some evidence of dilatation of the right ventricle and general signs of circulatory feebleness, as seen in the coldness of the hands and feet, local œdema and tendency in some cases to venous thrombosis. Murmurs systolic in rhythm are heard often at the four characteristic points in the cardiac area, but the most distinctive of these is the loud bruit heard over the pulmonary artery at the inner end of the second left inter-

space, and variously attributed to dilatation of the artery and to mitral insufficiency.

A continuous murmur or humming sound is heard at the base of the neck; this is the *bruit de diable*, heard over the lower part of the jugular vein on the right side. The condition of the blood will be dealt with under Diagnosis.

There is sometimes marked irritability of temper, hysterical symptoms, and in a very small percentage of cases signs of sinus-thrombosis or optic neuritis may manifest themselves.

The urine is pale and abundant, but otherwise normal, and as a rule there is no fever. The course of the disease varies, but generally it rapidly yields to treatment by iron. Relapses are uncommon when the administration of the drug is continued for several weeks after the re-establishment of the menstrual function.

Diagnosis.—As a rule there is seldom any difficulty at first sight: the sex and age of the patient, the absence of the menstrual discharge, and the presence of anæmia of a pronounced type, with the characteristic green tinge of the skin, and white sclerotic in a well-nourished patient with normal temperature, are all characteristic. The disease most likely to be mistaken for chlorosis is severe anæmia caused by hæmorrhage from a *gastric* or *duodenal ulcer*, whose existence is not suspected by the patient. An examination of the stools settles the diagnosis.

Early tuberculosis may cause mistakes, but a careful survey of the chest and abdomen, with thermometric observations, and an examination of the sputum, will prevent the error.

Kidney-disease is at once eliminated by an examination of the urine.

From all other forms of *anæmia* save one chlorosis is easily differentiated by an examination of the blood. The exception is the anæmia of malignant disease, which, however, does not occur at the chlorotic age, and which has, moreover, its own symptoms of wasting, &c.

The constant characteristic of chlorotic blood is the absolute diminution in the hæmoglobin; but, more than this, there is always a decrease in its amount out of proportion to the mere deficiency in the number of red blood-corpuscles. In other words, each individual corpuscle is poorer in hæmoglobin, and consequently the colour-index must be always less than 1. Thus the number of corpuscles may be diminished to 50 per cent., whilst the amount of hæmoglobin may be only 25 per cent. of the normal, giving a colour-index of ·5. The corpuscles, however, may be but little diminished in number, their average, according to Gulland, being about 3,800,000 in 1 c.mm. They are smaller in size and paler, blood-plates are very numerous, poikilocytosis, except in severe cases, is not marked, leucocytosis is absent, but there may be a slight

increase in the lymphocytes. Ebben has shown that there is increase of the fatty constituents both in the serum and in the erythrocytes.

In the anæmia of malignant disease, nucleated forms of red cells and leucocytosis are not uncommon, and in a doubtful case these points would exclude chlorosis.

Prognosis.—This is invariably good; the disease is never fatal except in the presence of a complication like sinus-thrombosis. Relapses are common, but very seldom when the iron-treatment has been pursued long enough.

Treatment.—'Iron' sums up the treatment; and notwithstanding the enormous list of organic iron salts, which is being extended by new compounds almost daily, there is no evidence that these are in any way superior to the older and better-known inorganic preparations like the sulphate, scale preparations, and reduced iron. The daily intake of iron in the food necessary for a healthy individual is not more than $\frac{1}{8}$ grain, but much larger amounts are necessary in the treatment of chlorosis. Blaud's pills may be given up to twelve or even twenty per day. A pill containing 2 grs. reduced iron and $\frac{1}{8}$ gr. arseniate of iron, thrice daily, may be substituted after three or four weeks. The iron-treatment should be continued for at least three months. Attention to the amount and quality of the food, and every change likely to improve the general health, as free open-air exercise, &c., are not to be neglected.

CHOLANGITIS. (See under GALL-BLADDER.)

CHOLERA ASIATICA

This is an acute disease, capable of being transmitted from man to man, produced by a specific microbe which causes violent purging, with rice-water evacuations and collapse.

Etiology and Pathology.—Endemic in India for many centuries, the disease was unknown in Europe till 1829–1830, since which period smaller or greater epidemics have appeared at irregular intervals during the century, and from Europe the disease has been introduced into America at different periods. The specific microbe is the comma bacillus discovered by Koch in 1884. It is a curved flagellated rod, about half the length of the tubercle-bacillus, but thicker; the older forms are more bent than the younger, and sometimes they are attached to each other in the form of the letter S, or in corkscrew fashion. They are found in pure cultures in the evacuations, and in the intestines of cholera-subjects, and in drinking-water, to which the disease has been traced. They have

been cultivated outside the body in various media, and though easily destroyed by drying and heat there is good reason to believe that under suitable conditions they may preserve their vitality in the soil for many months or years. The source of the disease is in all epidemic cases the bacillus, which has found its way into drinking or washing water, or into food, from the evacuations of cholera-patients, or from the stools of persons coming into close personal relations with cholera-patients. That it can in some rare instances be *air-borne* is possible, but in the vast majority of cases it is generally accepted that the disease is *water-borne*. The bacillus will not multiply in distilled water, though it continues to live for a few weeks in this medium ; and it increases rapidly in numbers on moist soiled linen, where it has abundance of oxygen. Flies act as carriers of the disease, and always play an important part in polluting milk, &c. The great epidemics from east to west have always spread along the lines or routes of the caravans, or amongst the pilgrims along the highways of human travel ; sometimes its distribution corresponds with the direction of the rivers, and innumerable instances of its being carried by infected ships are recorded. It is most active in its spread in warm and moist seasons, winter weather often checking an epidemic, which will break out again on the advent of early summer. It is rare in high altitudes, and flourishes always in the low-lying parts of the infected district. Even in severe epidemics, only a small proportion of the people may become infected, so that a considerable percentage are probably immune; and it is remarkable how seldom physicians and nurses are attacked. Debilitated persons and alcoholics are prone to suffer ; from their contact with the soiled linen, washerwomen are very prone to catch the infection ; and it seems probable that one attack confers immunity for a considerable time, and an entire community becomes immune for a few years after a severe epidemic. Often during epidemics a type of condition known as *Cholerine* is observed ; this, like *choleraic diarrhœa* so-called, is probably a mild abortive form of the disease, with symptoms resembling cholera nostras.

Symptoms.—The symptoms of cholera come on after an incubation-period varying from 40 to 160 hours. Often there is a premonitory diarrhœa for twelve to forty-eight hours, with slight colicky pains. The onset of the disease is marked by violent diarrhœa, the motions being copious and watery, presenting after the first few evacuations a colourless or turbid appearance like the water in which rice has been boiled ; the evacuations are alkaline, and on standing deposit flakes consisting of epithelial *débris*, mucus, and the specific bacilli. Very large quantities of this rice-water liquid are passed at short intervals without pain or tenesmus. Intense

thirst soon develops and vomiting sets in. This, like the diarrhœa, is copious and frequent, and attended with little mechanical effort. Collapse, restlessness, cardiac weakness, coldness and blueness of the extremities, with a drop of 6° F. or more in the axillary temperature, rapidly supervene. The face becomes pinched, the eyes sunken, the voice hoarse and feeble, the skin moist, ashy, cold, and shrivelled; dyspnœa occurs in paroxysms, the respirations being quick and shallow; the breath is icy cold, and the pulse almost imperceptible. In this, the *algid* stage of cholera, though the patient's body feels to the touch as cold as that of a corpse, the rectal temperature may record 103° or more. Severe cramps or tonic spasms set in, affecting the arms, abdominal muscles, and the calves of the legs; and though the vomiting continues, or ends in retching or hiccough, purging may cease from paralysis of the intestinal tube. The urine becomes scanty, or is entirely suppressed, and the salivary secretion is arrested; but the skin is covered with perspiration.

The algid or collapse stage is often fully developed within eight hours of the onset, and it may terminate in as many hours in either death or reaction; but sometimes it continues for sixteen or thirty hours, during which time the patient remains in a conscious, though drowsy or apathetic condition, which in fatal cases passes into coma. In rare instances death has supervened before purging has had time to occur. These cases have been described as dry cholera or *cholera sicca*.

The *reaction* stage sets in, in cases which recover, with a general improvement in all the symptoms; but there may be no urine secreted for twenty-four to forty-eight hours. The pulse becomes perceptible and gradually gains in strength; the respirations become slower; the dead cold of the extremities gradually passes off, and the skin becomes normal; the rectal temperature falls; vomiting, purging, and retching cease, and the agonising cramps disappear, the patient in very favourable cases falling asleep and awaking with much of his danger passed. Some of the cases relapse, and in a recurrence of the algid stage a fatal issue occurs.

The *typhoid* stage of cholera supervenes after the algid in a variable percentage of cases, and especially in the intemperate. In these examples of the disease, instead of the algid stage ending in speedy reaction, the patient passes into a low feverish condition, with great prostration, restlessness, dry tongue, vomiting, anorexia, and in many instances with evidence of uræmia, as seen in a scanty highly albuminous urine with casts. Death may follow, after coma has set in, about the fifth to the tenth day, but if the patient survives longer than this he generally recovers, though slowly. Thus the entire duration of the attack may be as short as a few hours, or as long as a fortnight or more. Various roseolar or erythematous

eruptions have been noticed during the stage of reaction and during the typhoid stage.

Complications, as parotitis, gangrene of the genitals and of the mucous membrane of the mouth, diphtheritic affection of the mucous surfaces, pneumonia, and pleuritis are occasionally observed.

Morbid Anatomy.—The *post-mortem* changes are not distinctive, and they vary exceedingly according to the stage of the disease at which death occurred. If in the early stages, the thick condition of the blood in the veins, and the empty state of the arteries and of the intestinal tract, with an airless and bloodless condition of the lungs and a remarkable dryness of the serous membranes, are most noticeable. When death occurs during reaction, the brain, lungs, liver, and kidneys show signs of congestion, and the small intestines may exhibit softening, ulceration, or diphtheritic exudation upon their mucous surface. Rigor mortis sets in early and lasts long, and the muscular rigidity is intense in degree, sometimes causing rupture of muscles. The only distinctive pathological characteristic is the presence of the comma bacillus in the congested intestines.

There is much uncertainty about the explanation of the phenomena of cholera, whether these are to be attributed to the presence of toxins manufactured by the bacillus, or to the mere extraction of fluid from the blood during the growth of the bacilli in the intestines. The extent to which this dehydration occurs may be realised by the consideration of the fact that a gallon of the cholera evacuation may contain a little over half a drachm of dried deposit. Parkes found four grains in one pint. The dehydration of the blood explains the presence of cramp, thirst, and suppression of urine, air-hunger, shrivelling of the face, &c. The bacilli are never found in the blood or organs outside the intestinal tube, and very seldom in the vomited matter.

Diagnosis.—*Severe* cases of cholera are not likely to be mistaken for any other affection, but there is a close resemblance between severe types of *English cholera* so-called and the milder forms of true cholera, and the symptoms may be almost identical, though in English cholera the stools are always more or less bile-stained. The microscopical examination of a flake of mucus from the bowel, spread out on a cover-glass, dried, and stained with fuchsin, if the cultivation be pure, will settle the diagnosis in a few minutes; but in all cases where there is any doubt about the identity of the bacillus, owing to the presence of other organisms, the flake should be incubated for twenty-four hours on peptone-broth. Such a culture will also give the so-called cholera red reaction upon the addition of a little strong sulphuric acid, and under skilled bacteriological methods the agglutination-test may be applied.

Arsenical poisoning and poisoning by antimony and perchloride of mercury have been mistaken for Asiatic cholera, but the symptoms on examination should not cause any real difficulty ; and, moreover, the poison is easily detected in the vomit, evacuations, or in the urine. More difficult are the examples of ptomaine-poisoning which may follow the eating of poisonous mussels, tinned fish, and fungi, but the history of the case almost always renders the diagnosis clear.

Prognosis.—The mortality all round is over 50 per cent., and in some epidemics may reach 75 per cent. during the first weeks of the outbreak. It is greatest in the very young and very old, in subjects labouring under cardiac and renal disease, in pregnant subjects, and amongst the intemperate. Those cases where the diarrhœa stops without improvement in the other symptoms, and where there is much lividity and prolonged suppression of urine, seldom recover. The prognosis is hopeful in severe cases where the rectal temperature falls as soon as symptoms of reaction set in ; when hyperpyrexia (which is, however, a rare complication) occurs, death is certain to follow.

Treatment.—Prophylaxis is of more importance than measures for the cure of the disease. As cholera is most frequently conveyed in water used for drinking and washing purposes, the water-supply should be the first consideration in all cases. Boiling thoroughly destroys the bacillus, and all water, even for washing purposes, should be boiled ; but of greater importance is the prevention of the pollution of the water-supply. The contamination is almost always brought about by cholera-discharges finding their way through the soil or, by means of sewage, into wells, streams, or reservoirs ; and in this connection it is to be remembered that the bacillus is found in the motions of patients seven weeks after the attack. All motions should be disinfected promptly by chlorinated lime or other antiseptic, and soiled linen and clothing must be treated in the same way. The bodies of dead victims should be cremated early or buried deep in the soil, with a layer of lime or other disinfectant inside the coffin. The most scrupulous cleanliness in regard to food, ventilation, and other sanitary matters should be enforced, and rigid isolation should be insisted upon. The ingestion of over-ripe or of unripe fruit, and of all food in a state of incipient decomposition, must be forbidden. In India, excellent results have been obtained by adding permanganate of potassium to the water in the wells.

Inoculation of attenuated cultures of the comma bacillus, followed in five days by injections of exalted or virulent cultures, as first carried out by Haffkine, has proved highly efficacious in protecting individuals during an epidemic. The protection lasts for about one year. Recently Haffkine

has recommended the injection of pure cultures of the bacillus as a preventive in the presence of an active epidemic.

All authorities agree as to the wisdom of treating promptly every attack of diarrhœa which occurs during a cholera-epidemic, but remedies are useless when given with the view of diminishing the secretion from the bowel, once the disease has become established. Intestinal antiseptics, with the possible exception of calomel, are worthless; and the best method is to wash out the bowel with large quantities of a warm solution of tannin (2 oz. to 1 gal. water) by means of the long tube. It is maintained that by gentle massage over the cæcum the fluid can be made to reach the stomach. Normal saline (6 drms. sodium chloride and 2 drs. sodium bicarbonate to 1 gal. water) may be injected in a similar manner; but better results are obtainable by injecting large quantities into the subcutaneous tissue, with the view of making up for the great amount of fluid lost from the blood. In desperate cases the liquid should be injected into the veins. In the algid stage, the warm bath may be resorted to with benefit, or a hot pack may be used; alcoholic stimulants are usually clearly indicated in free doses, and sometimes oxygen inhalations are serviceable. Cramps, when violent, demand hypodermics of morphia; but opium by the mouth for the relief of pain, or with the view of checking diarrhœa, is generally valueless, since it is usually not absorbed. The only drug of any proved value being calomel (which often relieves the vomiting), it may be employed in the early stages only, in full doses (10 grs.), by the mouth, in conjunction with the tannin-injections; but its routine administration, especially during the collapse stages, is fraught with considerable danger; and opium in this stage must also be condemned.

The symptoms arising in the stage of reaction and in the typhoid stage must be treated upon general principles, and drugs must be then avoided or used with the greatest caution. The only food permissible during the attack is milk diluted with barley-water, beef-tea, clear soup, or meat-juice; and even these should be withheld during the early period, ice alone being permitted to control the vomiting and assuage thirst.

CHOLERA NOSTRAS

This is an acute gastro-enteritis, ushered in with vomiting and purging, and exhibiting many of the collapse-symptoms of true cholera. It is known as Choleraic Diarrhœa, and English Cholera, and seems to be identical with Cholera Infantum.

Etiology and Pathology.—The main point in its pathology is the absence of Koch's comma bacillus, otherwise the majority

of the cases could not be differentiated from true cholera. The morbid changes observed are those found in acute catarrhal inflammation of the stomach and bowels; sometimes the solitary glands and Peyer's patches and the mesenteric glands are found enlarged. It is most frequent in the summer months, and the specific microbe, if there be one, has not yet been isolated. Some authorities believe that the symptoms are produced by a definite change in the virulence of the bacillus coli communis, the result of the ingestion of an irritant; others attribute the disease to ptomaine-poisoning, which various kinds of bacilli have been supposed to originate in the intestinal contents.

Symptoms.—The attack sets in suddenly, with violent purging, generally, but not always preceded or accompanied by vomiting. Severe griping pain is constant, and always more prominent than in true cholera. The motions are at first highly offensive and bile-stained, but gradually tend to become like those seen in cholera; they possess a scalding or irritating character, which excoriates the anus and intensifies the sufferings of the patient. As the amount of watery discharge increases, the clinical symptoms of collapse become more marked; cramps and thirst are prominent, the features get pinched, the eyes sunken, the skin shrivelled and cold, the temperature in the axilla falls below normal, and the urine diminishes in amount. Death may occur from the severe collapse, the patient passing into coma; but this is only likely to take place in cases of severe ptomaine-poisoning in the adult, or when the disease attacks infants or enfeebled old subjects.

In *cholera infantum* the symptoms are identical; they appear generally during hot summer weather in *bottle-fed infants*, the chief etiological factor usually present being milk contaminated by various micro-organisms which have already started fermentative or other changes in the liquid. The attack commences suddenly, with severe vomiting, soon followed by incessant purging. The stools are at first offensive and brown or greenish in colour; they gradually change to an inoffensive alkaline fluid like the rice-water stools of cholera, though often some bile continues to appear in them. As a rule, the prostration and collapse increase more rapidly than in the adult, and in twenty-four to thirty-six hours the typical '*hydrocephaloid state*' may be reached, in which the infant lies unconscious, with sunken eyes, pinched features, depressed fontanelles, a dry red tongue, a high internal temperature, with a cold ashy skin, imperceptible pulse, retracted head, and respiration of the Cheyne-Stokes type. Death may occur before the expiration of thirty-six hours; more frequently the end is postponed till after the third day.

Recovery sometimes follows, even when the coma has been complete, and after convulsions have appeared.

Diagnosis.—As already stated, the main point is the exclusion of Asiatic cholera, and this is only thought of in the presence of an epidemic of that disease. Poisoning by arsenic or other mineral irritant is excluded by the history and by testing for the poison, if there be suspicious circumstances present. Investigation of the milk and other food-supply is of great importance in the case of children ; but the diagnosis of cholera infantum is not likely to be associated with difficulty, owing to the rapidity of the onset, the intensity of the vomiting and purging, the speedy collapse, and the elevated rectal temperature.

Prognosis.—In cholera infantum this is always serious, and very grave in the ill-fed and neglected. In the adult the prognosis is generally good, unless in ptomaine-poisoning caused by eating a meal of mussels or decomposing shell-fish, pork, sausages, &c.

Treatment.—Owing to the rapidity of the onset of the enteritis, any attempt to remove the morbific cause by purging only increases the collapse. In adults the best practice is to give as little food as possible : a little iced milk or iced water to allay thirst is all that is needful as far as nourishment is concerned ; but the symptoms of collapse must be promptly met by stimulants : small quantities of brandy, whisky, or iced champagne should be given at brief intervals. Vomiting may be relieved by smart counter-irritation over the epigastrium, and severe cramps and colic by a hypodermic of morphia. The plan of drenching the patient with vegetable astringents, chalk, opium, &c., is to be condemned. Warmth to the surface is of vital importance. The ideal treatment would be the administration of an intestinal antiseptic ; but all these are valueless in acute cases. Much good can be obtained, however, in some cases by flushing out the intestines with warm saline solution in copious amounts, and it may be necessary to administer this hypodermically.

In *cholera infantum* the same principles must be adhered to. The milk-supply must be immediately changed, and little food of any kind given for twenty-four hours. Small quantities of barley-water and beef-juice meet the requirements at first, followed afterwards by egg-albumin and peptonised milk in small amounts. Stimulants are indicated in the great majority of cases at an early stage, and 15–20 mins. of brandy may be given in barley-water every hour. The only drug of any value is calomel in small and frequently repeated doses ; ½ gr. may be given every hour by placing it upon the tongue with 1 or 2 grs. sugar, and washing it down with a teaspoonful of iced water. Flushing the bowel with saline solution, and giving this liquid hypodermically, may be resorted to

in desperate cases. After the collapse has been got over, a small dose of castor-oil has always a highly beneficial action, and, if possible, a wet-nurse should be obtained for the future feeding and cow's milk abandoned. Where this is impossible, the milk should be sterilised and occasionally peptonised, and beef-juice given from time to time.

CHOREA

This is a spasmodic disease generally occurring in youth, characterised by jerky involuntary movements, with loss of coördinating power in the voluntary muscular actions, some muscular and mental weakness, and often associated with or preceded by rheumatism and endocarditis. It is often described as Sydenham's Chorea, and popularly designated St. Vitus's Dance.

Etiology and Pathology.—Most cases occur between the ages of seven and fifteen; girls are affected nearly three times more frequently than boys, and the coloured races are almost exempt. The disease is rare after twenty. Heredity, temperament, mal-nutrition, overwork in school, the exanthemata, various reflex irritations, and other causes have been credited as etiological factors in a small percentage of cases; but the chief causes about which there can be little doubt are rheumatism, fright, and pregnancy.

Rheumatism is undoubtedly closely associated with chorea: in a considerable number of cases it has preceded the attack; in a slightly larger number it has occurred at the same time or subsequent to the chorea. Endocarditis is also intimately associated with both affections, and many authorities regard the valvular lesion which is so commonly met with in chorea as evidence of a previous rheumatic attack; but undoubtedly endocarditis sometimes occurs during the chorea without joint-manifestations. Those cases where chorea has followed scarlatina are accepted also as being of rheumatic origin, since the diplococcus rheumaticus is frequently associated with the scarlatina infection.

Fright has so frequently immediately preceded an acute attack of chorea that the causal relationship cannot be doubted. The writer was fortunate enough to witness personally the beginning of an attack of chorea in a healthy child within a few minutes after the tragically sudden death of her father. In the majority of the numerous recorded cases the symptoms do not appear for days after the fright or shock; this is probably because the first signs are overlooked. In the case just referred to, a medical man standing by could not satisfy himself that there were unmistakable choreic symptoms; but the case was uncommonly well marked within forty-eight hours after-

wards, and ran its typical course, during which a mitral murmur developed.

Pregnancy has an unmistakable association with chorea in a small percentage of cases, and it often leads to serious results. It appears generally before the third month, and in first pregnancies. Though it may recur during subsequent pregnancies, it never has been known to occur for the first time during a third or later pregnancy.

The pathological changes observed after death from chorea are not constant; the most frequent is the presence of endocarditis. Hyperæmic states of the central nervous system and embolic plugging of the small vessels, especially in the large basilar ganglia, have been often noted, but have been found entirely absent in other typical cases. The lesion, whatever it may be in chorea, is almost certain to be cortical.

The pathology of the disease has long been a vexed subject, but recent advances in our knowledge of the nature of acute rheumatism are gradually clearing up the difficulty. There can be little doubt about rheumatism being a disease of micro-organismal infection, and the diplococcus rheumaticus isolated by Poynton and Paine plays a most important, if not the entire, part in the rheumatic phenomena. These investigators produced choreiform movements in rabbits by the intravenous injection of this organism; after death it was found in the pia mater, and they also found the micrococcus present in the motor cortex in a fatal case of chorea. Others have cultivated the coccus from choreic patients, and have been able to produce symptoms allied to acute rheumatism in rabbits by injecting it. Moreover, the two observers named have produced malignant endocarditis by injecting the rheumatic micrococcus.

The old theory that chorea was the result of showers of minute emboli into the vessels of certain parts of the brain must be now abandoned; all the above-mentioned facts point to the newly discovered micro-organism as the chief agent, and hence the growing tendency to regard the disorder as cerebral rheumatism. The only difficulty in accepting this as a working hypothesis lies in facts like the one detailed upon the previous page, where the symptoms come on immediately after a fright. The nature of the action on the highest centres is probably paralytic, the microbe or its toxin in this way removing their inhibitory power over the lower cortical centres, which are then allowed to run riot.

Symptoms.—These come on in mild cases after a few days of restlessness or fretfulness, after which it becomes obvious that the child is unable to sit still. She soon begins to drop things from her hands as a healthy girl might do under severe mental emotion or nervous embarrassment; and this being

attributed to awkwardness, it is usual to find that punishment has been inflicted, which rapidly makes matters worse.

Irregular characteristic involuntary movements now become marked; they may involve the facial muscles, the hands, or the feet. When the first-mentioned are affected, grimaces of various kinds are produced which sometimes closely resemble those made voluntarily by a naughty child; the muscles of the mouth, eyelids, tongue, and jaws being seen to suddenly contract and relax in an irregular manner, unlike that seen in any other nerve-disease. The arm is also constantly involved, being abducted or jerked from the side and suddenly brought back again with shrugging of the shoulder; the wrist is extended and flexed, or the hand pronated and supinated, and the fingers flexed and extended alternately. The trunk and head-muscles may participate in these purposeless vagaries, the respiration being irregular, and even the vocal cords may join in the disturbance. The leg is affected to a less degree, but not less frequently, and an examination of the boots of nearly every choreic child will show, when inspected, marks of friction and wear never seen under other circumstances; the writer has noticed this in the absence of arm and facial involuntary movements, and it may be a valuable diagnostic aid. The movements vary in strength, frequency, and in range, from being so slight as to be perceived only by the trained eye to those of such violence as may knock the patient off his feet or jerk his body out of bed, or cause serious injuries to the skin, joints, or bony prominences. They are more marked under any mental or emotional excitement, and they generally cease during sleep; their distribution is irregular, but sometimes they are confined strictly to one side of the body, constituting *Hemichorea*; more frequently they are as irregular in their distribution as in their nature or degree, and they often change from one side to the other. Occasionally only one arm may be affected; but the muscles of the face and trunk, when involved, are always affected bilaterally.

The voluntary movements show *loss in coördinating power*, and in some cases this may be present before the onset of the purposeless, jerky, spasmodic, and involuntary movements. Thus, articles may be dropped from the hands by the premature relaxation of the flexors; or upon attempting to grasp an object groups of antagonistic muscles may be called into action, which render the achievement awkward or impossible. If the patient be directed to put out the tongue, there is generally a delay, after which the organ is shot out with suddenness and retracted as rapidly, or it may be sometimes caught between the teeth by the sudden snapping of the jaws together. Speech is seriously impaired in this way, and the handwriting is defective, sometimes proving marked incoördination when there are only slight involuntary movements. As a rule, how-

ever, the ataxy and involuntary manifestations are mixed up together, and the incoördination is made prominent by an attempt to correct or neutralise some involuntary movement.

Weakness of the affected muscles is common, rarely extending to paralysis ; but it may be so marked as to justify in some cases the term used by Gowers of *Paralytic chorea.* The arm is most markedly affected, but sometimes it may be of the paraplegic and sometimes of the true hemiplegic type ; it may be present early, or not till after the attack of chorea has subsided.

The electrical reactions of the muscles may be normal, but sometimes they are increased, and the same may be said of the reflexes ; the knee-jerk occasionally presents choreic characters, the extension is prolonged and associated with choreic movements in the toes. Sensory disturbances are slight ; headache and pain in the affected regions are, however, sometimes noticed, and there may be blunting of sensation. Fever is absent, but erythematous rashes supposed to be of rheumatic origin are often present.

Mental weakness, or a varying degree of psychical disturbance, is present to some extent in most cases ; generally it is slight, but it may be severe, and may end in total loss of mind-power. It is important to remember that it bears no relation to the severity of the case, and must always be taken into account in the prognosis. In one type of case the mania is so marked that the term *Chorea insaniens* has been given to it, and sometimes the degree of psychical disturbance is so great as completely to overshadow the chorea and lead to the mistake of making a diagnosis of insanity. Epilepsy may supervene in rare cases.

The *heart* is very frequently involved in chorea, and much interest attaches to the association. Murmurs are found in more than half of the cases. These may be owing to a previous rheumatic attack, in which cases there will be evidence of hypertrophy. There is nothing to support the theory that the murmurs are the result of irregular contractions of the papillary muscles causing mitral regurgitation. They are doubtless in some cases caused by anæmia ; but endocarditis is an almost constant feature in chorea, as it is in rheumatism, and in nearly all the fatal cases it is found on *post-mortem* examination. The remarks already made upon the pathology of chorea explain the meaning of the endocarditis : it must be regarded as a local manifestation of the rheumatic infection caused by the micrococcus. The left side of the heart is always involved in these cases, the mitral valve suffering and the aortic escaping. There may be no murmur audible during the chorea, but it may be heard long after convalescence ; and hence it is evidently possible that an endocarditis may be present during the attack of chorea which does not betray its

existence at the time save in rapidity of the pulse. Pericarditis occasionally is present. Rheumatism may have preceded the attack, or appeared at its onset, or developed during its height, or it may follow it later on, after complete convalescence: hence the explanation of the systolic murmur heard at the apex, and, as in rheumatism pure and simple, a marked murmur heard during the attack may pass off and leave no trace of valvular mischief behind it, or it may remain as evidence of a permanently damaged valve. The arthritic manifestations may be very slight, or they may be overlooked during the chorea, and the writer has witnessed their place being taken by the characteristic erythematous rash of peliosis rheumatica; and after this purpuric eruption had faded, joint-pains and swelling returned again. Occasionally typical fibrous nodules may be felt under the skin.

The duration of the attack varies exceedingly: in mild cases it is often over in six weeks. In the type of cases called *Chorea gravis*, where all the symptoms are greatly intensified and where the ceaseless movements interfere with rest, feeding, and sleep, death may occur in less than a couple of weeks. Other cases of moderate severity may pass into *Chronic chorea*, and this is more liable to occur in adults. The paralytic cases are very slow to recover. Recurrences are prone to take place, and it is not unusual to find patients who have had three or four attacks separated by intervals of variable lengths, the subsequent attacks being often milder than the original. Pregnancy is very liable to cause recurrence in those who suffered from an attack during their first pregnancy, and many of these patients have had attacks during childhood and severe worries in the early months of gestation.

Diagnosis.—In the great majority of cases an error is scarcely possible, but *Friedreich's ataxia* has been confused with it; this is a congenital affection with a remarkable family history: the nystagmus, lateral curvature, talipes, peculiar gait, scanning speech, and absence of knee-jerk are all characteristic.

Athetosis, which may follow an infantile hemiplegia, with its peculiar mobile spasm, sometimes affecting both sides of the body, may be mistaken for chorea, but only in the absence of a history, as it always follows an early cerebral hæmorrhage or birth-palsy. The movements are slow, and there is marked spasticity and rigidity; one movement slowly passes into another, constituting a sort of cycle which is characteristic. A form of athetosis, also known as *Post-hemiplegic chorea*, occurs in adults after hemiplegia. The hand is the part most commonly involved, and the spasm affects chiefly the interossei, when the fingers and wrist are alternately flexed and extended in an irregular rhythmical manner. The history of the hemiplegic attack settles the diagnosis in these rare cases.

Chorea spastica is a condition sometimes confused with chorea: it occurs after infantile hemiplegia, and is often associated with mental weakness; but the tremor resembles that of *disseminated sclerosis*, which latter affection should be easily differentiated from chorea by the oscillations or tremors being only produced on an attempt to perform some movement (they are *intention*-tremors), and nystagmus and scanning speech are marked features.

Hemiplegia may be mistaken for paralytic chorea; but even in the best-marked cases of this latter disease some characteristic choreic movements can almost always be detected, and the paralysis never affects the face. Choreic paralysis of one arm in a child is always gradual in its onset, and, as a rule, all cases of slow paralysis of one arm in a child are choreic.

Poliomyelitis anterior acuta may also simulate paralytic chorea; but the marked wasting, the reaction of degeneration, and the rapid or sudden onset are characteristic.

Paramyoclonus multiplex is differentiated by its lightning-like clonic contractions or shocks, which are generally symmetrical, and do not at all resemble voluntary movements; they cease or are inhibited during the performance of voluntary acts which always exaggerate choreic movements.

Hysteria may simulate chorea closely, as may be seen in the cases which are produced by imitation. Girls are nearly always the subjects of this form; the movements are often rhythmical, and always more isolated and sudden than in chorea.

Mania can hardly be mistaken for chorea; but maniacal chorea may be easily mistaken for simple mania where the choreic movements are slight or overshadowed by the active delirium. Mania is rare at the typical choreic age, and its almost incessant or continuous rambling speech is more prominent than that seen in chorea insaniens.

The rarer forms of chorea are differentiated by their characters described in the succeeding article.

Prognosis.—This is generally good in children, the all-round mortality not exceeding 2 per cent.; but the prognosis is much less favourable in adults, and it is serious in pregnancy, where the mortality rises to 25 per cent., chiefly from dangers attending abortion, delivery, and cardiac complications. When chorea attacks adults, it is more likely to assume the chronic form; in children, when death occurs, it most frequently happens in first attacks. There is always some danger of permanent mental weakness in those cases where the psychical disturbances are well marked, regardless of the amount or intensity of the choreic movements; but, as a rule, recovery is to be expected. Even in tedious cases of post-choreic paralysis the prognosis is good, as return of power may be expected in every case unless there be some organic

brain-lesion, which is extremely rare in children. Epilepsy may be a permanent sequela in those rare cases complicated by convulsions.

Treatment.—Only general principles need be here referred to. The writer has dealt with the details in his 'Dictionary of Treatment,' pp. 144–149.

Rest of body and mind is essential, and all excitement must be guarded against by isolation from other children; school-work should be given up at once when the symptoms are noticed. Feeding and sleep must be carefully attended to and constipation avoided.

Arsenic is the drug upon which most reliance is to be placed; but to be of any use it must be given in full doses for considerable periods. A seven-year-old child may commence with 3 mins. of Fowler's solution thrice daily after food, and this may be increased gradually till 12 mins. be taken in each dose. Neuritis and pigmentation of the skin have been recorded, but they very rarely follow, even when the drug has been pushed. In the chorea of pregnancy abortion should not be induced; bromides are harmful, but arsenic is beneficial. In the severe cases of chorea gravis, where the intensity of the movements threaten life, chloral may be freely given to induce almost continuous sleep for considerable periods, and hemlock may be combined with it. Even chloroform narcosis may be demanded by the urgency of the symptoms.

Recurrences must be guarded against after complete recovery. School-work should be carefully regulated; all sources of reflex irritation, as the presence of worms, errors of refraction, adenoids, anæmia, should be sought out and corrected; and frights must be guarded against as far as this is possible, as they are very liable to lead to recurrences.

CHOREA, HEREDITARY

This affection is also known as Chronic Progressive Chorea, Chronic Adult Chorea, Senile Chorea, and Huntington's Chorea.

It is a progressive disease, generally of hereditary origin, coming on between the ages of thirty and forty years, with slow irregular involuntary movements, incoördination, difficulty in speaking, a swaying gait, and gradual loss of mental power.

Etiology and Pathology.—The disease has no association with rheumatism or with Sydenham's chorea. Several members of one family may suffer, and the condition has been traced through many generations.

The anatomical changes which have been found point to a chronic pia-arachnitis, with a degenerative sclerotic encephalitis and atrophy of the convolutions, the cortical neurons suffering early.

Symptoms.—These begin slowly, generally in the hands, with movements somewhat like common chorea ; but they gradually become more irregular and disorderly, and much slower than those of chorea, till all the muscles in the body are involved. Facial grimaces, gesticulations, and ocular spasms are present ; irregular movements of the tongue and laryngeal muscles cause speech to be very difficult, and writing becomes impossible ; though for a long time voluntary movements may be performed with good effect, and during their performance the involuntary action is in abeyance. The gait becomes unsteady and swaying like that of a drunken person, and finally, after many years, walking becomes impossible from the gradual development of a high degree of spasticity in the legs. The mental condition slowly and steadily deteriorates ; suicidal tendency is often present ; but finally a state of chronic apathy supervenes and ends in dementia.

The disease is incurable, but the patient may live on to senility. In some cases of chronic chorea presenting the above characters no hereditary history is obtainable. The onset may be after the age of fifty and up to seventy years, and occasionally improvement sets in ; these cases have been grouped by some writers under the term *Senile chorea*, and if they are not identical with the hereditary form they are nevertheless not associated with rheumatism ; but there may be no mental decay accompanying senile chorea, and sometimes recovery has been known to follow.

Electrical chorea, first described by Dubini, occurs mostly in Northern Italy, and is suspected to be a malarial or other endemic parasitic intoxication. It is nearly always fatal, affecting all ages, and beginning with feverish symptoms, head-, neck-, and back-ache. Soon spasmodic involuntary movements set in in the arm and leg on one side ; these resemble choreic movements, but they are shorter, come on more suddenly, and resemble a series of galvanic shocks. They cross over and affect the opposite side, and the muscles soon show wasting ; hence the toxic agent probably acts first upon the motor cortex, and secondarily upon the large cells in the anterior cornua of the cord. Paralysis soon follows, and epileptiform convulsions are not rare. Death may occur inside a week or it may be postponed for several months, and no treatment has any action in arresting the malady.

The disease is to be distinguished from *Bergeron's electrical chorea*, which is a variety of the ordinary chorea of childhood, with smarter, more rapid, and sudden shock-like movements ; it is probably of hysterical origin, and yields to ordinary chorea treatment.

Habit chorea is the term sometimes applied to Habit or Histrionic Spasm, which is described under its own name. It is a totally distinct affection from ordinary chorea.

CHROMIDROSIS

Is the name given to the rare condition in which the secretion of the sweat or sebaceous glands is coloured. Nothing is known definitely of its pathology ; but the undoubtedly genuine cases occur most frequently in hysterical or markedly neurotic women about the menstrual period and in association with habitual constipation. The presence of indican in the urine in chronic constipation renders it probable that some derivative of indol may be absorbed from the intestinal tract, and in its elimination may cause staining of the sweat or sebum in those cases where these secretions are stained brown, black, or blue. The condition is only of interest from its extreme rarity, and from the ease with which it may be artificially produced by hysterical patients.

After extreme cleanliness, free purgation, and treatment directed to the hysterical or menstrual affection, the phenomenon usually rapidly disappears.

Hæmatidrosis, or bloody sweat, has unquestionably been seen, but only in hysterical subjects, in whom it may appear as a vicarious form of menstruation. Red sweating is a different condition, which has been traced to microbes, especially upon the hairs of the axilla. Green sweat is a common condition found in workers in copper, and various drugs have been at times found to colour the sweat and milk, after being absorbed from the stomach ; thus rhubarb colours the sweat a faint yellow after repeated administration.

Urea may be eliminated by the sweat-glands ; and as it crystallises upon evaporation of the watery solvent the surface of the body may appear as if covered over with a fine white pigment. The condition has been seen in uræmia and cholera, and is known as *uridrosis*.

CHYLURIA

Or, as it is also called, Hæmatochyluria, is the name given to the condition in which chyle appears in the urine.

Symptoms.—Save the presence of the chyle in the renal secretion, there may be no other symptoms or signs in many cases, and were it not for the characters of the urine the patient would be unaware that there was anything wrong with him; but repeated attacks cause much weakness, especially if there be blood in any amount, and there may be backache and depression. The urine often coagulates in the bladder, even when no blood is present, and the coagula may cause retention and serious local disturbances by obstruction of the urethra.

The urine when voided is opaque, milky-white, or pinkish, and thick. On standing, coagulation speedily occurs; but the clot soon contracts and the liquid is found to separate into three layers. The upper one is a thin layer of cream showing numerous small fat-globules and granules with fine coagula. The intermediate stratum, which makes up the bulk of the specimen, contains the clot, with much fine molecular fatty matter, leucocytes, and erythrocytes. The lower layer contains whatever blood is present as a pink precipitate which becomes redder after some hours, numerous small clots, epithelium, and numerous immature filariæ. The urine may be blood-red when passed, owing to the number of red blood-corpuscles present. There is always much albumin. If the fresh urine be shaken up with ether, it becomes normal in appearance; there is generally a lower s.g. than normal. In some cases, however, the urine is not opaque and milky, but clear and free from fat-globules—a condition known as *lymphous urine*. Filariæ may be found in the morning or night urine, though they may be more abundant in the night or in the morning secretion. Urinary casts are seldom found, as there is usually no structural alteration present in the renal organs themselves, except in some cases where a varicose condition of the lymph-channels has been noticed.

After a few days the patient may pass normal urine, but the chyluria returns from time to time for many years, and in some cases it may be permanent for long periods. In most cases a varicose condition of the inguinal lymphatics may be felt in the groins.

Etiology and Pathology.—The condition is very rarely seen except in those who have resided in the tropics, but occasionally instances have been reported of chyluria in subjects who had never been out of the British Isles. All ages and both sexes are liable to it, and it is common in all tropical climates.

The pathology of the affection has been carefully worked out, and the disease has been found in most instances to be owing to the presence of adult filariæ which live in the lymphatics. Several species of filariæ have been found in the human subject, and there is still some uncertainty about the identification of some of them; but there is little doubt that chyluria and elephantiasis are the result of a filaria to which in its mature form the name *Filaria Bancrofti* has been given. This is a hair-like or filiform worm, the female measuring up to five inches in length, while the male is about half this length. These parasites live in the lymphatics of the trunk and extremities, the females pouring into the lymph-stream many thousands of young filariæ, which pass along the lymphatic vessels and glands, through the thoracic duct, into the blood. They are in this immature stage thread-like,

measuring in width the diameter of a red corpuscle, but about forty times longer—*i.e.* about $\frac{1}{50}$ inch ; as many as five hundred may sometimes be counted wriggling about actively in a single drop of blood, each surrounded by a delicate sheath which is larger and much longer than the worm itself.

These young filariæ are absent from the blood in the daytime ; but towards evening begin to appear, and increase in number till midnight, when they gradually diminish, and finally disappear about seven or eight o'clock A.M., finding their rest in some unknown region of the body till the following evening. The parasite is known in this young stage as the *filaria sanguinis hominis nocturna*, and its intermediate host is a female mosquito, which takes it into its stomach by sucking the blood of a filarious subject. From the body of the mosquito the worm in a matured and sheathless form finds its way into drinking-water, by which route it enters the human stomach.

There is a worm whose embryos, which are much smaller, only appear in the blood in the daytime ; it is known as *filaria sanguinis hominis diurna*, and Manson believes that its mature form is *F. loa*, a parasite which infests the subcutaneous tissues of the negro. It is worth remembering that *F. nocturna* was found by Mackenzie to change its habits and appear in the daytime if the patient reversed his sleeping and working hours.

That chyluria can occur without filariæ is certain ; but the cases are extremely rare, and probably owe their origin to the same causes as operate in non-parasitic chylous ascites—*i.e.* to rupture of a lymphatic vessel in connection with the urinary tract.

Filariæ produce chyluria by blocking the main lymph-stream either with masses of the immature embryos or with quantities of the ova, which the mature parasite may under some conditions expel prematurely, or the parent worms may be the cause of the obstruction. The chyle and lymph-stream being blocked in the thoracic duct, the liquid, in order to reach the blood, must pass through the anastomosis of the lymphatic system of the duct with the lymphatic system draining the upper part of the body. According to Manson, the current flows backwards through the pelvic, scrotal, and inguinal lymphatics, to travel finally by the lymphatic vessels in the abdominal walls to the upper parts of the body. The lower vessels become varicose, and a rupture of one of these will produce chyluria, if in the renal or vesical system ; or if in the scrotum, the condition known as *Lymph-scrotum* ; or if in the tunica vaginalis, á *Chylocele*. The lymph-scrotum and the varicose tumours so constantly found in the groin in filariasis and chyloceles, are all easily identified by the use of the hypodermic needle, which draws

out an opaque milky-white fluid containing the characteristic fatty molecules or globules, and sometimes filariæ. Elephantiasis is probably produced in the same way, only here the ova, which are immaturely expelled (before they have had time to become embryos), are probably the cause of the blocking of the lymph glands and vessels in the affected extremities or genitals.

Diagnosis.—The characters of the albuminous urine, its colour and clotting, the revelation under the microscope of the fatty granules and the embryo filariæ, and the history of a residence in a tropical or sub-tropical climate, establish the diagnosis easily. The varicose inguinal glands may be mistaken for a hernia, and the chylocele for a hydrocele, but the hypodermic syringe settles the diagnosis in both instances.

Prognosis.—Filariasis is compatible with perfect health, and may last during a long lifetime without giving any trouble. Frequent attacks of chyluria may cause considerable weakness, and inflammatory or erysipelatous attacks may supervene upon the condition of lymph-scrotum, which may ultimately pass into elephantiasis, or vesicles consisting of varicosities of lymph-vessels may form upon the surface, and these, when they burst, discharge for a long period quantities of lymph, the loss of which slowly undermines the patient's strength. When the chyle discharges into the peritoneum or pleura, the chylous ascites or pleural effusion constitutes a serious drain upon the general nutrition.

Treatment.—No drugs are known which will destroy the parent or embryo filariæ in the blood or lymphatic vessels. In an attack of chyluria the patient should at once be put to bed, and the amount of liquids restricted, milk and fats being prohibited; the pelvis should be raised. Manson states that a single draught of milk, by causing albuminuria and clotting, will prove at once whether the rupture has closed, and until this is certain the patient should be kept in the recumbent position. Chylocele should be tapped and treated as a hydrocele, and lymph-scrotum should only be excised when elephantiasis has supervened. The varicosities and glands in the groin are better left alone or supported by a suitable bandage; but when they become so painful and enlarged as to cripple the patient they may be excised, though sometimes elephantiasis has followed. The preventive treatment in filarious districts will consist in the boiling and filtering of all drinking-water.

ELEPHANTIASIS ARABUM

Though chiefly of surgical interest, this disease is here referred to because of its relationship to chyluria. It is a very common affection in all tropical countries, and may be regarded as an

advanced or exaggerated condition of lymph-scrotum. The legs and scrotum are the parts usually affected; but it may involve other parts, as the mammary glands and female genitals, and even the face. The condition follows attacks of lymph-angitis with feverish symptoms, and erysipelatous inflammation often accompanies the inflammation of the lymphatic vessels. The attacks follow at irregular intervals, each attack leaving the affected part more infiltrated, till after some years the leg presents the appearance implied by its name. The hypertrophy of the skin and subcutaneous tissue may reach to an enormous degree, so that a scrotal tumour may weigh as much as an average human being, or more, or the leg may increase to thrice its girth, the hypertrophied tissue hanging in folds separated by deep sulci. The surface is dark in colour, warty, and rough, and fissures or deep ulceration may result from the retained secretion between the folds. Manson explains the supervention of elephantiasis on the filarious condition by the theory that the mature *F. nocturna* in a lymphatic vessel in the leg gets injured, and instead of pouring her many thousands of young active *embryos* into the lymph-stream, from which they pass easily into the blood (being only of the width or breadth of a red blood-disc), she showers instead her immature *ova*, which are blunt and larger. These are unable to traverse the small vessels and glands, and cause blocking of the entire lymphatic system in the limb, upon which supervenes inflammation leading to the characteristic changes. This interesting hypothesis explains also the general absence of the filariæ in the blood in these cases, as the embryos cannot traverse the obstructed glands.

Diagnosis.—The condition must be differentiated from chronic cases of *phlegmasia alba dolens*, which are non-parasitic and which follow parturition.

Prognosis.—The condition is seldom fatal unless when abscesses and erysipelas supervene, or when the enormous mass becomes the seat of gangrenous patches.

Treatment.—When the scrotum is involved the testicles may be dissected out and the entire tumour removed, with excellent results; but in the great majority of leg-cases all that can be done is to pay the most scrupulous attention to cleanliness, and to apply the gentle pressure of a pure rubber or elastic-webbing bandage, and to see that the limb is kept elevated.

CIRRHOSIS.

(See under LIVER; BRIGHT'S DISEASE; BRONCHIECTASIS, &c.)

COCCYDYNIA, or COCCYGODYNIA

Is the name given to pain in the region of the coccyx produced by neuralgia of some terminal branches of the sacral plexus, or of the fifth or sixth sacral trunks. The term is sometimes applied in a wider sense to the painful condition which follows injury to the lower end of the sacrum or fracture of the coccyx, generally produced during labour. The pain is aggravated by sitting or defæcating, and sometimes walking increases it also. It is often most rebellious to treatment, but some cases yield to rest and ordinary anti-neuralgic remedies. Simpson's operation consisted in the subcutaneous section of the coccygeal muscles, while Nott excised the coccyx.

COLIC

Though still sometimes called Enteralgia, this is but a symptom. The name is applied to pain which is produced by irregular contractions of the muscular apparatus of the intestinal tube. When the pain is the result of spasmodic contractions of the muscular walls of the biliary passages or of the ureter, the condition is spoken of as hepatic colic or renal colic.

Etiology and Pathology.—Intestinal colic, as above defined, may have its seat in the small or large bowel, the latter is more frequently involved in the typical condition, and the term is generally applied only to those cases where there is no organic obstruction. More or less colic accompanies the action of every cathartic when given in large doses, and there can be little doubt but the cause of the pain is the same in those cases produced by indigestible foods like pork, fungi, pastry, and those containing ptomaines : it is produced by the powerful contractions of the muscular tube brought into play with the view of driving onwards the irritating contents. In all probability the harmonious working of the circular and longitudinal fibres is disturbed, and the circular fibres, by contracting spasmodically, imprison the gas or other contents in small portions of the tube, which become distended ; and the longitudinal fibres then being brought into action in vain, the pressure is increased and the terminal nerve-fibres suffer. As the spasm of the circular fibres passes off, relief from pain follows. A similar event takes place whenever the bowel becomes obstructed from any cause, as in obstinate constipation, hernia, intussusception, obstruction from bands, stricture, &c. ; but, as a rule, the pain is only severe when the obstruction is a recent one, except in those cases where a slowly growing obstruction of the colon may suddenly bring on severe colic

by an effort of the tube to drive hardened fæces through the strictured part.

In *lead colic* probably other causes are at work, especially as constipation is the rule.

Symptoms.—An attack of colic comes on suddenly after a brief warning of uneasiness or distension. The pain is generally centred about the umbilicus, from which it may radiate in various directions. It is of a severe tearing, wringing, or boring character, as a rule, relieved somewhat by pressure, though in some cases there may be transient tenderness. The patient turns upon his face or leans over some support, so as to induce pressure upon the abdominal muscles. A still more characteristic feature is the paroxysmal nature of the pain, which may stop as suddenly as it commenced, and often the patient is conscious at the moment of relief of the displacement of gas or flatus in the bowel ; nausea or vomiting may accompany. The paroxysm may not be repeated, but an attack of colic generally consists of a series of painful spasms of variable duration, with intervals of complete absence of suffering.

The abdomen may be distended ; more often it is retracted. There is a normal temperature and moist skin and slow pulse, though in rare cases there may be an increase in the pulse-rate and slight feverishness. The aspect of the patient is in marked contrast to the anxiety and collapse seen in peritonitis, though nervous subjects may appear somewhat frightened. The attack lasts longer when there is constipation, and generally relief follows if diarrhœa sets in, or when purgation has been induced.

In *infantile colic* the symptoms are similar ; the attacks are caused by the irritation of coagulated cow's milk in the intestines, or by the accumulation of gaseous products of intestinal indigestion. In *children* colic commonly follows gorging with nuts, unripe fruits, gooseberry-hulls, cherry-stones, &c., and rapidly yields after the bowel has acted.

Colic always ends satisfactorily and safely, but there is, especially in neurotic individuals, a tendency to recurrences, although the condition has nothing in common with neuralgia.

Diagnosis.—As a rule this is easy, but caution is always necessary, even in apparently obvious cases, since formidable inflammatory and obstructive lesions of the bowel may be ushered in by colicky symptoms.

Acute obstruction of the bowel is soon followed by nausea, with severe and obstinate vomiting, which may become speedily stercoraceous ; no flatus is passed per rectum ; the abdomen becomes tympanitic and distended, unless the obstruction be situated high up near to the stomach. The clinical picture rapidly deepens, even in the brief intervals between the spasms, the countenance becoming anxious and pinched,

the pulse thready and rapid, and symptoms of collapse, with thirst and dry red or brown tongue, supervene.

Perforation of a hollow viscus and acute *peritonitis* from any cause, as *appendicitis*, usually are followed speedily by a grave general condition of collapse, which can hardly be mistaken for colic. The local signs are also characteristic—marked tenderness of the distended immovable abdominal muscles and the diffuse character of the pain, which is markedly aggravated by even the gentlest pressure, are obvious.

Lead colic is recognised by the marked retraction of the abdomen, the slow hard pulse, obstinate constipation, the blue line along the gums, and the presence of lead-palsy.

In *renal colic* the pain is centred in one flank, from which it radiates downwards towards the groin and into the testicle and penis; there is generally frequent desire to micturate, and blood or gravel may appear in the urine.

Hepatic colic begins with very sudden violent pain under the right costal arch, radiating upwards into the chest and backwards into the right shoulder. There may be a rigor, with elevation of temperature, vomiting, and profuse sweating, and generally there is marked tenderness over the region of the gall-bladder. Jaundice often, but not necessarily, presents itself after some hours.

Appendicular colic, so-called, may be mistaken for ordinary intestinal colic, but there is generally a history of similar attacks, commencing always in the region of McBurney's point where there is much localised tenderness, and drawing up of the thigh. Since these cases are really mild examples of appendicitis, in some attack severe inflammatory symptoms will eventually appear.

Uterine colic occurs generally at the menstrual period, with other symptoms pointing to uterine disturbances, as discharge, hæmorrhage, &c. When the Fallopian tube is involved, the pain, which is central in the former case, is found to be lateral and associated with ovarian tenderness, and is generally followed by a clear or purulent discharge from the vagina.

Gastralgia comes on when the stomach is empty, as can generally be demonstrated by percussion, and the pain is confined to the gastric region and seldom radiates so low as the navel.

Movable kidney sometimes simulates colic, but as a rule the pain is found to have started in the loin or flank, is of a severe dragging character, and has probably followed some muscular movement, and when intense is associated with scanty or blood-coloured urine; palpation will, moreover, reveal the displaced organ.

Treatment.—The first indication is the relief of pain; but seldom will it be necessary to resort to morphia, hypodermically or in large doses by the mouth, and never need opiates

be given in the infantile form. A large hot poultice, or an indiarubber bottle or hot stupe, to the abdominal wall, and the administration of a carminative like oil of peppermint, anise, cinnamon, or cajuput in doses of 5 mins, with 20 mins. spirit of chloroform or teaspoonful doses of tincture of ginger, generally afford speedy relief. In very severe cases a hot bath and morphia hypodermically, or a whiff of chloroform may be needed. Pain being relieved, the bowel should be emptied by a large warm enema, to which a teaspoonful of tincture of asafetida has been added, and an aperient should be given as soon as the diagnosis is certain. One purgative having advantages over every other in the important quality of harmlessness should be invariably selected, and this is castor oil. It may be given with a little laudanum or with any carminative, or with a small dose of brandy.

In infantile colic, 1 to 2 mins. of oil of anise, followed by a teaspoonful of castor oil, is all that is needed.

COLITIS

Inflammation of the colon is met with in three well-marked forms: (1) *Simple catarrhal*; (2) *ulcerative*; (3) *membranous*.

Etiology and Pathology.—*Simple catarrhal* colitis, also called simple mucous colitis, is but a name for catarrhal enteritis when the process is confined to the colon. It is used also by some writers loosely as a synonym of diarrhœa (which see). There is inflammatory action with redness and swelling of the mucous membrane and increased secretion identical with the condition seen in the visible mucous surfaces when the seat of inflammation, though in simple enteritis affecting the colon this may not be very evident after death. In the chronic form thickening is always to be met with. The causes of the primary form are the ingestion of improper foods or irritants, or the action of ptomaines or bacterial products manufactured in the intestines, especially in hot weather. The various micro-organisms found in the motions probably play no part in the causation of the disease.

Symptoms.—The symptoms are those of ordinary catarrhal enteritis, the chief feature being diarrhœa, which in the acute cases comes on suddenly, associated with more or less pain and griping. The motions are fluid and often devoid of ordinary fæcal matter, consisting of mucus with some fluid blood, which may be passed separately or, as is most frequently the case, it is intimately mixed up with the mucus. As a rule, there is nothing like the tenesmus seen in dysentery; straining may be absent, though the motions may be very frequent, unless the rectum becomes involved. The pain may sometimes be found to correspond with the position of the colon,

and to be relieved after the evacuation; tenderness can generally be elicited over the sigmoid flexure. There may be feverishness, thirst, nausea, or vomiting, and the tongue is generally covered with a white fur.

The attack may pass off in a few days; but it may recur, and a chronic course is not very unusual which leads to anæmia, wasting, and much general and mental depression, or hypochondriasis. When the disease is secondary to such conditions as Bright's disease, pyæmia, enteric or other infectious fever, it generally runs a severe and rapidly fatal course. The explanation of the pain is supposed to be enterospasm, the result of a reflex from the irritated bowel, and this can be almost demonstrated sometimes by the hand placed over the colon and sigmoid during severe pain, when the intestinal tube will be found contracted into a firm cord.

Diagnosis.—*Enteric fever* is differentiated by its slower onset, higher temperature, and characteristic fetid pea-soup motions, in which mucus to any amount is very unusual. Widal's reaction or the appearance of the eruption will settle the diagnosis.

Malignant disease of the upper part of the rectum may closely simulate the chronic form of colitis; the amount of mucus streaked with blood in the motions, which are passed with considerable pain and straining, is liable to mislead, but a rectal examination will settle the diagnosis in every case where the growth is within the reach of the finger.

Ulcerative colitis is differentiated by its slow onset and by the characteristics described in the succeeding article.

Dysentery is eliminated by the absence of a history of residence in the tropics, and by an examination of the stools, which are also generally less numerous. The dysenteric motion is very scanty: it contains serum, blood, mucus, and pus, without any fæcal matter, and the amœba may be found in it; there is generally much pain and tenesmus.

Hyperpurgation caused by drugs like gamboge, jalap, &c., is easily eliminated by the history; but the wicked system of practical joking by giving croton oil has often led to mistakes, and some cases of homicidal poisoning by arsenic and corrosive sublimate have been overlooked because of their resemblance to simple colitis.

Prognosis.—In acute cases this is good, as few end fatally unless there be severe ptomaine or other irritant poisoning causing the condition; but in chronic cases, especially in neurotic subjects, the prognosis as regards duration is unsatisfactory, as such cases may last many months.

Treatment.—This resolves itself into the management of the diarrhœa. The patient should be put to bed and fed upon small quantities of milk and arrowroot. Thirty-grain doses of bismuth carbonate suspended in fresh mucilage should be given

every few hours, and when the diarrhœa is profuse, astringents are indicated; but it is not advisable to drench the patient with catechu, kino, &c., which upset the stomach and may not reach the colon at all. Tannalbin in 30-gr. doses passes through the stomach unchanged; it is to be preferred to the ordinary vegetable galenical preparations, and may be given in wafers with bismuth, and a little opium if necessary may be combined with it.

Compound decoction of aloes sometimes acts like magic in a single dose of 1 oz., but if it fail it need not be repeated. Castor oil is the safest drug for the constipation which is liable to follow. Tenesmus, where present, may be relieved by a small enema of starch and laudanum.

COLITIS, ULCERATIVE

This is a specific inflammation of the colon, tending towards the formation of numerous ulcers, which may destroy a considerable extent of the mucous membrane down to the muscular coat.

Etiology and Pathology.—The inflammatory action starts in the submucous layer. The ulcers are not follicular; according to Hale White, they are probably the result of a micro-organism, and there may in severe cases be only islets of unaffected membrane left here and there throughout the entire length of the colon. The disease is not to be confounded with tubercular, dysenteric, or typhoid ulceration. It occurs in middle life, is common in asylums, and is often associated with chronic Bright's disease.

Symptoms.—These consist mainly of intense colicky pain and severe diarrhœa resembling dysentery, though tenesmus and straining are not generally prominent. The motions are foul, and often contain small scybalæ. They are dark and liquid, and often blood-clots or liquid blood and small shreds or sloughs are present, but there is not the abundance of mucus seen in the simple variety of colitis. Constipation sometimes alternates with the diarrhœa, as in the common form of intestinal tubercle, but the motions are generally much more frequent and painful. In severe cases great prostration and fever are usually marked.

The onset is slow in most cases, and the disease when established is generally chronic and progressive, wearing the patient down by the production of vomiting, anæmia, and emaciation, expedited by fever and sometimes by perforation, peritonitis, or hæmorrhage.

Follicular colitis is always secondary to some other affection and is not recognisable during life, and is hence only of pathological interest.

Diagnosis.—The greatest difficulty is found in the elimination of *dysentery*. The absence of a history of a residence in

the tropics is important; the motions are not dysenteric, but present the lienteric character. The disease only attacks, as a rule, middle-aged subjects, and these often are suffering from chronic renal affection. The numbers of the motions, though great, never reach those met with in some cases of dysentery, nor is there such marked tenesmus generally observed, except when the ulceration extends towards the anus, when a digital examination may demonstrate its presence, and this will also differentiate it from *cancer of the rectum*.

In a case recently seen by the writer, the condition of the rectum strongly suggested ulcerative colitis, but the history and obvious presence of tubercle in the lymphatics and lung settled the diagnosis as one of extensive *tubercular ulceration* of the lower end of the great bowel.

Prognosis.—This is always grave, most patients succumbing inside three months. One apparently hopeless case under the writer's notice, which had lasted nearly six months, made a permanent recovery, and there could be no doubt about a very considerable extent of the mucous surface of the colon being destroyed.

Treatment.—Rest in bed, milk-diet, with thin arrowroot, and animal jelly, gelatin, or isinglass, should be prescribed. Astringents and antiseptics by the mouth probably only hasten the fatal termination, and certainly do not add to the patient's comfort. The best procedure is to flush out the colon daily, or oftener, with a copious enema of saturated, warm, boric-acid solution, after which every other day a quart of solution of nitrate of silver ($\frac{1}{4}$ gr. to 1 oz.) should be injected, and kept as long as possible by the patient. Colotomy may be indicated in some cases where the pain and distressing diarrhœa are uncontrollable by drugs, and from the opening on the right side the colon may be flushed, by allowing the antiseptic solution to flow out through the anus.

COLITIS, MEMBRANOUS

This is an affection of the colon, characterised by the passage from the anus of mucous casts of the interior of the bowel. The disease is also known as Muco-membranous Colitis and Membranous Enteritis.

Etiology.—Osler regards the disease as a secretion-neurosis of the colon. Four out of every five subjects of this affection are females, and nearly all of them are neurotics suffering from gastric symptoms and often from uterine troubles. The disease is rare in children. A secondary form is met with in the dead-house in association with conditions like the last stages of pyæmia, Bright's disease, cancer, enteric fever, pneumonia, &c.

Symptoms.—The casts may be tubular and may measure many inches in length; they appear more frequently in whitish, stringy, rope-like masses, and they consist of dense mucus, devoid of fibrin, though of transparent, homogeneous structure, in which, however, may be found epithelial and other cells, and fæcal *débris*.

The symptoms, in addition to those belonging to the neurotic depressed state, and the irritable dyspepsia, consist of attacks of severe abdominal pain in the region of the colon, accompanied with some tenesmus, nausea, and unusual mental depression, coming on almost always when the patient is obstinately constipated. The pain is greatly increased during the patient's attempts at defæcation, which are not always successful, and purgatives even of the mildest type do not usually diminish the suffering, though they may be effective in causing an evacuation. The attacks, with longer or shorter intervals during which the motions may be almost normal, generally last for years; the duration of the individual attacks is usually one or two days, but they may be prolonged for as many weeks, and there may be severe attacks of pain without the passage of any membranes.

The disease is very seldom fatal, but tends to reduce the patient's strength and weight notably, and most of the victims pass gradually into confirmed invalids, to whom life becomes a burden. Dilatation of the stomach and colon is frequently met with in such chronic cases, and enteroptosis and nephroptosis are common.

Arthritism is also a marked feature in severe colitis, either showing itself late in the progress of the case by a recrudescence of old joint-troubles, or by its early appearance, or by the history of joint-pains, rheumatism, gout, and rheumatic arthritis.

Diagnosis.—There can scarcely be a difficulty about this, unless in first attacks, when indigestible vegetable material may be mistaken for the stringy casts. In one case, the writer saw repeatedly the motions of a patient supposed to be suffering from this affection for months; the flakes and supposed casts were often passed, sometimes in little balls, and sometimes in stringy masses. The microscope revealed these to be portions of orange-pulp, which the patient (a male) confessed that he had been in the habit of bolting daily in large amounts. There was often much mucus and some painful diarrhœa, from occasional attacks of simple colitis. Appendicitis is sometimes caused by colitis; sometimes colitis is associated with appendicitis, and, as pointed out by Caley, it often simulates appendicitis. This is more true of the chronic catarrhal than of the membranous variety.

Prognosis.—As already stated, the tendency of the disease is to end in hypochondriasis, or hopeless invalidism; but cases occasionally get well spontaneously, and do not recur if the

neurotic condition passes off. More frequently a vicious circle is established, and, as stated by Caley, neurasthenia predisposes to colitis, and colitis induces or aggravates neurasthenia.

Treatment.—This is most unsatisfactory, and, as a rule, purgatives only aggravate the condition. The main hope of recovery lies in the successful treatment of the neurotic condition by improved hygiene and the alteration of the patient's environment. Pain should, if possible, be relieved by other means than the use of opiates, and large enemata of warm boric solution, or of olive oil, generally afford relief. Hale White urges the opening of the colon on the right side, and the keeping open of the artificial anus for at least six months, with the view of giving the colon complete rest.

Caley has recently given a classification of the several types of colitis which affords a bird's-eye view of the subject. The following table is reprinted from his valuable article in the 'British Medical Journal,' June 1906, since it seems to the writer the first successful attempt to form order out of chaos.

I. Catarrhal, or simple mucous, colitis.
   (1) Acute:
      (*a*) Primary (that is, primary infective inflammation of the colon).
      (*b*) Secondary (that is, secondary to other morbid states—for example, specific fevers, toxæmias, Bright's disease).
   (2) Chronic:
      (*a*) Irritative type.
      (*b*) Atonic and spastic types.

II. Membranous, or muco-membranous, colitis.

III. Parietal colitis and pericolitis (including 'sigmoiditis,' pericolitis sinistra, and perityphlitis).

IV. Ulcerative colitis.
   (1) Endemic (tropical dysentery).
   (2) Epidemic (for example, 'asylum dysentery').
   (3) Sporadic:
      (*a*) Primary, 'simple,' or 'idiopathic.'
      (*b*) Secondary (that is, secondary to general diseases or to local affections of the lower bowel—for example, stricture).

A very rare form of colitis, known as *colitis polyposa*, has been observed by Pope and by Carey Coombs, and the interesting pathological changes are reproduced from Coombs's case in the 'Medical Annual' for 1907, by Robert Hutchinson. The colon was found studded over thickly with numerous small polypi; there was no ulceration or evidences of past ulceration present. The polypi were found to consist of an overgrowth of round cells derived from the connective-tissue cells of the submucosa.

COMA

Complete loss of consciousness is produced by a variety of causes which shut off or suspend the functions of the cerebral convolutions. Coma is therefore but a symptom of many different diseases or conditions, the great bulk of which are grave; and as these are treated under their own names it is only necessary to group them here. Milder forms of coma are spoken of as *somnolence* and *stupor*; but when the word 'coma' is used, it is intended to imply a condition of complete unconsciousness, from which it is impossible to arouse the patient. In stupor and somnolence the reflex action in the limbs is present always, and sometimes in increased form; in coma it is lessened, and in profound coma it is generally abolished. The condition need not be here described; suffice it to say that it presents itself in its typical form in the condition of chloroform narcosis, and its chief features are referred to under 'Cerebral Hæmorrhage.'

Coma, more or less profound, is met with in the following conditions:

1. Cerebral apoplexy, whether the result of hæmorrhage, embolism, thrombosis, concussion, or laceration of the brain from violence.
2. Inflammatory conditions within the cranium, as meningitis, encephalitis, abscess, sinus-thrombosis, &c.
3. Cerebral affections of a non-inflammatory nature, as tumour, cerebral syphilis, epilepsy, infantile convulsions, heat-stroke, general paralysis, multiple sclerosis; perhaps, for convenience, hysterical coma may be included in this category.
4. Narcotic poisoning, as by alcohol, opium, chloral, chloroform, ether, and other general anæsthetics, carbonic acid, carbon monoxide, and coal-gas.
5. Kidney-diseases affecting the renal structure, when the state known as uræmia follows, or calculi blocking both ureters.
6. Diabetes in its later stages, from the accumulation of β-oxybutyric acid.
7. Hepatic diseases in their terminal stage, as cirrhosis, obstruction of the bile-ducts, and acute yellow atrophy.
8. The poisons produced during the progress of the infectious fevers, as typhus, typhoid, small-pox, and scarlatina; septicæmia, pyæmia, pernicious malaria, pneumonia, sleeping sickness, the hyperpyrexia of acute rheumatism, erysipelas, &c. In this category may be also included asphyxia from whatever cause, which ends in the retention of carbon dioxide in the blood, and the coma that has been seen to follow violent muscular exertion, independent of heat-stroke, and the coma following prolonged exposure to intense cold.

The condition known as *Coma vigil* is a variety of coma generally seen in typhus and typhoid fever, in which the patient lies with his eyes wide open, though he is completely insensible to all going on around him, and his face is devoid of expression, though he may continue to mutter.

Syncope is excluded from the above list, though for a brief period there is absolute unconsciousness owing to acute anæmia of the brain.

The differential diagnosis of the most important varieties of coma will be found detailed under 'Cerebral Hæmorrhage.'

CONCUSSION OF THE BRAIN

This is a term used to include the symptoms which supervene after a general shaking of the brain, the result of violence. All visible injuries, lacerations, or hæmorrhages are excluded in the following brief description of concussion. Minor cases often come under notice when the original cause has been forgotten or overlooked. There are three well-marked stages in all severe cases: First the stage of *collapse*, in which there is more or less unconsciousness, which may last from a few minutes to hours, or days; the surface of the body and the extremities are cold, the conjunctival reflex is absent, the pulse is very weak and thready, and the sphincters may be relaxed. After the return of consciousness the period of *reaction* is generally ushered in by vomiting and some restlessness, the skin gets warm, the face flushed, the pupils contract, the pulse becomes full and bounding, and sleepiness supervenes, which may last for some days. Headache follows; but gradually the patient passes into the stage of *convalescence*. In those cases of great severity where the stage of reaction passes into meningitis or cerebritis, there is probably always some laceration of the brain-substance or punctiform hæmorrhages. Mild cases, following falls on the feet or buttocks, or diffused blows of soft substances, may be associated with no unconsciousness at all, or at most with a slightly dazed feeling, which rapidly passes off. Bennett, in Allbutt's 'System,' draws attention to the importance of these minor attacks, which may afterwards come under the care of the physician for headache and various mental peculiarities. When the injury has been direct, as from a blow, the after-symptoms consist of rapidity of pulse (which is characteristic in being compressible on the slightest touch of the finger) and a mental mood of irritability and antagonism. The after-symptoms in those cases where the injury was indirect, as from a fall on the buttocks, where the shaking is presumed to have affected the parts above the cerebro-spinal junction, are as follows: pain at the back of the head, heaviness amounting to lethargy

or mental apathy, and a slow pulse-rate, sweatings, and great muscular wasting.

Diagnosis.—The main point in all cases of severe concussion is to watch for symptoms of asymmetry in all the stages. Thus, a weakness in one limb or in one side of the body, or in the face, will indicate paralysis, which at once puts the case out of the category of simple concussion; conjugate deviation and other ocular phenomena are also of value in the exclusion of simple concussion.

Prognosis.—This is always serious till the diagnosis renders it probable that no brain-laceration complicates the concussion. In simple concussion, though there may be sequelæ, as mental excitability, &c., following, still, complete recovery may be usually expected.

The gravity of the prognosis deepens with the prolongation of the period of unconsciousness, and death may occur from the collapse or shock. Prognosis in all cases is much influenced by treatment.

Treatment.—This is apt to be overdone. In the stage of collapse the patient should, as a rule, be left severely alone, in a condition of absolute repose, with his head slightly lowered. Horsley, in very severe cases, advises warm water to the head by means of Leiter's tubes, and others advise purgation by croton oil or calomel; but it cannot be a mistake at this stage to leave the patient at complete rest, even refraining from shouting into his ear to test his consciousness. All stimulants are injurious; but should the heart be very weak, ammonia may be applied to the nostrils. In the stage of reaction everything will depend upon the continuance of the enforced repose of body and mind. Calomel may now be given with advantage, ice should be applied to the scalp; and if the symptoms of reaction get formidable, leeches should be applied behind the ears or to the temples, but the opening of a large vein in the arm is certainly more efficacious when the blood-pressure is high.

Judicious starvation for the first two or three days, followed by a spare milk-diet, is the best course, and under no circumstances should animal food—not even beef-tea—be permitted for many days, or even weeks in bad cases; and when solid food is given for the first time, its effects should be watched, and a return to milk insisted on if headache occurs or the pulse-rate be quickened. Alcohol must be forbidden for months, and the return to the upright position only permitted after the pulse has become normal. There is perhaps no other condition in which prolonged physical and mental rest is so essential as in the convalescing stage of brain-concussion. In railway concussion there is difficulty in carrying out the rest-treatment, as the patient is continually worried about legal matters, and many of the cases fail to improve till the question of com-

pensation is settled. In these cases there is always an element of neurasthenia, and the mental impression produced by the accident may induce a marked neurosis, totally independent of any brain or spine concussion. To this condition the term Railway Brain or Railway Spine is applied, according to the prominence of the cerebral or spinal symptoms.

CONCUSSION OF THE SPINAL CORD

This may be produced by falls from a height or from general shaking, caused in railway collisions. There is great difficulty in determining at first whether there is any gross lesion of the cord, but if paralysis or other marked spinal symptom or sign rapidly follows, the case is evidently not one of simple concussion.

There may be no symptoms whatever for several days after the accident, when gradually or suddenly meningeal or paralytic symptoms may appear, with optic neuritis, bladder disturbances, and death may result without any obvious lesion of brain or spine being found.

Diagnosis.—The great bulk of these cases, however, may be regarded as examples of traumatic neurasthenia (see under 'Neurasthenia') induced by the mental impression caused by the accident; and the difficulty of separating the hysterical or neurasthenic elements from malingering is sometimes insuperable, as all the symptoms are purely subjective. A careful examination will often reveal attempts at deceitful exaggeration of the reflexes and ocular symptoms. The mixture of sensory and motor disturbances, especially if the latter be monoplegic, and if contractures be unattended by wasting and abnormal electrical reaction, is almost certain to be hysterical, and limitation of the field of vision for white and coloured light may accompany this, with increase of reflexes, tremors, and general paresis.

Upon the other hand, evidence of lateral sclerosis, marked muscular atrophy, and interference with the lumbar centres in the cord, or signs of compression-myelitis or angular curvature, will demonstrate that the case is one of irreparable injury to the cord and not one of traumatic neurasthenia or simple concussion.

Prognosis.—In simple spinal concussion recovery is generally rapid and complete. In traumatic neurasthenia the prognosis is good, but the progress towards recovery is always slow. Many cases show no tendency to improve till the worries incidental to litigation are removed. Any tendency to hypochondriasis, as ascertained from the past history of the patient, or evidence of mental disturbance in the family record,

introduces an element of gravity as regards the duration of his symptoms, and even in respect to his final recovery.

Treatment.—In acute cases, or in patients seen immediately after the accident, absolute rest in bed is all that is required. Traumatic neurasthenia must be treated upon the general principles laid down for the relief of neurasthenia; but if there be much backache or pain, it will be wise to compel the patient to take complete rest in bed, during which time, after the lapse of a period sufficient to relieve any strain or laceration of ligaments or muscles which might possibly be caused by the injury, counter-irritation by blisters or the cautery may be tried. Horsley condemns bromides, alcohol, and travelling, all so frequently prescribed in the chronic stage, and recommends in every case resort to the Weir-Mitchell treatment by rigid isolation, massage, and over-feeding.

CONSTIPATION, or COSTIVENESS,

Is the name given to the condition in which the fæces are retained longer in the bowel than they should be in the normal state of health. Only the subject of chronic or habitual constipation is here dealt with.

Etiology and Pathology.—The causes of constipation are numerous, but above all others in both frequency and importance, there is one which should never be forgotten: without a thorough appreciation of its powers for evil, a cure can hardly be expected. This factor is the habit of disregarding the call of nature, or of postponing the response to it till a more convenient season occurs.

In the normal condition the fæcal matter remains as a rule in the colon, at or about the sigmoid flexure, and it does not descend into the rectum till just before defæcation should take place. Its descent in the great majority of healthy individuals occurs once in the day, and very generally it takes place after the morning meal, probably synchronising with the establishment of digestion after the stomach's long night rest. The rectum, upon receiving the contents of the bowel above, is stimulated, and the result is the call of nature just referred to. If attention to this be postponed, the delicate nerve-mechanism becomes disturbed and the natural sensibility of the rectum blunted, because, instead of being empty at all times, save the few moments before defæcation, the rectum is constantly full of fæcal matter, whose tendency is to become drier and harder. After a few days, or weeks, this alarum-clock action of the rectum ceases, evacuation being accomplished by the force of the contraction of the muscular walls of the colon driving the hard contents of the paralysed or toneless rectum in front of the more recent feculent

material, the abdominal and other muscles assisting in the act.

In the normal healthy individual this descent of the bowel-contents into the rectum at a certain period of the day constitutes a sort of rhythm, and it is the re-establishment of this rhythm which must be the objective of all treatment.

Sedentary habits, abnormal conditions of the brain as in mental diseases, and of the spine as in paraplegia, of the pelvic nerves as in disease of the uterine appendages, mechanical difficulties as in narrowing of the lumen of the colon, or the pressure of a retroverted uterus or ovarian tumour—may cause constipation.

The improper use of purgatives is a potent factor in many cases, and this operates probably by destroying or disarranging the rhythm. The normal stimulus to peristalsis is the indigestible portion of the food, as the cellulose covering of the cereals and the fibrous residue of vegetables ; when fine flour takes the place entirely of whole meal, the irritating quality of the bran is absent, and constipation ensues. But of even more importance is the *quantity* of food taken. It is a most important and significant fact, that the great bulk of all constipated patients are very spare eaters, and that constipation is very rare in big feeders ; the surplus above what is needed and can be digested acts as a natural laxative. People who drink very little fluids are prone to suffer, and when the small quantity of fluid taken consists almost entirely of strong tea, the tannin still further tends to dry up the contents of the upper portion of the colon. Much has been written about the enfeeblement of the muscular layers of the colon ; but it is very doubtful if this is often a primary cause, unless in weak and bed-ridden patients, or in those recovering from long and exhaustive illnesses, and the same remark applies to weakness of the abdominal muscles, which, in the typically normal condition, play a subordinate part in the act of defæcation. Brunton lays stress upon the constipating effects of lime in the water used for drinking and cooking. Hawkins has recently drawn attention to a form of chronic constipation which is due to a *spastic condition* of some portion of the colon, associated with chronic abdominal pain, simulating in many instances appendicitis: the contracted colon may often be palpated through the abdominal wall. This form, which he designates as *spastic constipation*, is usually found in neurotic or neurasthenic subjects.

Symptoms.—The symptoms of constipation are obvious. They consist in the retention of the fæces or in the passage of dry or hardened motions ; but a condition is almost constantly overlooked which the writer finds to prevail with great frequency, especially in women. In this class of case the bowels may be moved daily more than once, and always at irregular

hours ; the motions are small, and an examination reveals the fact that the rectum is never emptied. The state of the lower end of the bowel in these cases bears some resemblance to the condition of the atonic and dilated bladder in advanced prostatic obstruction, though there is little or no tenesmus. It seems as if the evacuation were produced by the fæcal matter driven into the rectum simply displacing a small portion (its own bulk) of the drier fæces stored in the rectal cavity. This condition should always be suspected when two or more constipated stools are passed daily, and when hæmorrhoids are present.

The symptoms which supervene upon retention of fæces may be very slight, and apparently perfect health may be maintained for many days without any evacuation. Many persons have in their normal condition only one motion every second or third day, but such are always small feeders. As a rule, however, some feelings of malaise, abdominal uneasiness, depression of spirits, headache, and inability to perform brain-work are experienced ; though how much of these may be merely owing to the consciousness of the bowel having ceased to perform its natural office it is difficult to say, especially in neurotic subjects.

The local dangers are certainly not imaginary. Hæmorrhoids, ulcerations, fissures, bladder-troubles, uterine displacements, sacral or sciatic neuralgia, menstrual irregularities, colitis, and eventually intestinal obstruction may arise, requiring prompt intervention. The opposite condition of an exhausting diarrhœa with the rectum full of hardened fæces is not rare, and once the writer found the entire colon after death packed with scybalous masses tunnelled with openings through which an offensive diarrhœal discharge had found its way for several months ; this was in the case of an elderly religious fanatic who obstinately refused all treatment.

Treatment.—In the author's 'Dictionary of Treatment,' pp. 163–172, the principles and details of treatment are discussed at considerable length ; here only a few guiding rules need be mentioned. Drugs are harmful, unless used with the sole object of restoring the lost rhythmical action of the colon and rectum ; purgation, unless in acute or transient constipation, should never be induced. The physician should aim at assisting the patient to empty the rectum every morning after his first meal of the day ; when this can be accomplished without drugs the success is permanent. He should prescribe food which leaves a considerable indigestible residue, as whole-meal or brown bread ; wheaten or oaten-meal porridge ; abundance of vegetables ; fruits, as tomatoes, stewed prunes, figs, baked apples, and oranges in the form of marmalade or uncooked. Perhaps the best single article of diet is the boiled Portugal or Spanish onion ; and many cases of habitual

constipation may be permanently relieved by this vegetable without any drugs. Before dressing in the morning a tumblerful of cold, or warm, but not hot water should be prescribed, and the patient should be directed to go to the water-closet within a quarter of an hour after breakfast; but he should be told not to strain or use strong voluntary expulsive efforts, though massage of the abdominal muscles may be permitted. Fissures, hæmorrhoids, and prolapse are the frequent result of the fashionable advice that patients 'should solicit' an evacuation by prolonged bearing-down efforts.

If the motion does not take place after a short delay, a glycerin or soap suppository may be inserted and retained for several minutes; and should failure follow, a copious enema of tepid water must be used, the patient lying down on a rug on his left side, with his head low.

After a patient trial of this plan, a mild laxative pill containing 1 or 2 grs. extract of aloes with $\frac{1}{4}$ gr. extract of nux vomica, or a pill of 2 or 3 grs. extract of cascara, or 5 grs. of rhubarb pill-mass may be prescribed to be taken immediately before the evening meal. The physician must insist upon the intelligent co-operation of the patient, and impress him with the fact that the above doses will probably be too little or too great; if too little, the enema is to be resorted to at the stated morning hour, and the following evening two pills or one pill and a-half should be taken, or, if the effect has been a purgative one, only half a pill need be taken. In a few weeks the exact dose is thus accurately determined and the difficulty is on a fair way towards solution. Such a pill may be taken every evening for many years without harm. It may be necessary to alter the time for taking the laxative dose so that it will cause the evacuation at the required hour; and in order to accomplish this, it may be necessary to postpone the swallowing of it till bed-hour, or to administer the pill about the hour of the afternoon cup of tea. In cases where the morning hour is an impossible one for the evacuation of the bowel the evening may be selected, and the dosage timed accordingly about breakfast-hour.

In young intelligent patients who carry out instructions to the letter, the physician will often be able gradually to diminish the dose after a time till drugging becomes unnecessary.

Should the pill be forgotten, or from some irregularity in meals or other cause should it fail to act, the enema must be brought into use without waiting till next day. (This is the proper indication for the use of the enema or suppository in the treatment of chronic constipation.)

Other aids must not be omitted, as brisk exercise in the morning; and some of the various forms of apparatus devised for physical culture may be fitted up in the patient's bedroom. Massage or, as some recommend, a small cannon-

ball (7 lb.) covered with wash-leather may be moved over the colon, beginning above the right groin and causing it to travel over the direction of the colon towards the sigmoid as the patient lies upon his back before getting up for the day.

It may be necessary, before beginning the method outlined above, to give a brisk purge, or, if the colon or rectum be blocked, a large enema of olive oil or warm water. In some cases of acute or severe constipation the masses in the rectum must be broken up with the finger, or the handle of a spoon, or other convenient instrument.

Sulphur, liquorice-powder, rhubarb, and hosts of preparations containing other purgatives, are vaunted, but nothing acts so uniformly as aloes or cascara in habitual constipation. The liquid extract of this latter drug is especially valuable, owing to the facility with which the dose can be diminished or increased. Iron may often be combined with the aloetic pill in doses of 1 gr. of the dried sulphate, and some physicians attach importance to the combination of a small dose of powdered ipecacuanha-root; but as a rule the best adjuvans is strychnine or nux vomica.

CONVULSIONS

This term is usually applied to a series of purposeless muscular contractions which, though sometimes beginning in a limb or other portion of the body, spread over the entire voluntary muscles. When the convulsion is confined to any particular region it is generally termed a *spasm* (see also under 'Spasm').

Convulsions are in character either *tonic*, as in tetanus, where the contraction is continuous; or *clonic*, where the phenomenon consists of a series of rapidly alternating contractions and relaxations, such as are seen in epilepsy and eclampsia of puerperal or uræmic origin.

The practical or clinical division of convulsions would be into those which indicate organic brain-disease, and those which are functional in their nature; but this division is not always possible, as the pathology of many so-called functional affections is still obscure.

Organic brain-disease causing convulsions is generally situated in or near to the cortex, and is seen in its most typical form in the so-called focal or Jacksonian epilepsy, which, though beginning in the irritation produced in a particular motor centre, may spread rapidly to all the voluntary muscles in the body. Such local commencement is always indicative of organic brain-disease.

Meningitis, cerebritis, apoplexy, tumours, infantile hemiplegia, hæmatoma of the dura, abscesses, syphilis, softening,

and other organic cerebral lesions, may be the cause of general convulsions; the degenerative condition known as general paralysis of the insane, and any cause producing marked increase in the intracranial pressure, as chronic hydrocephalus, may give rise to general convulsions.

Various poisons, as those produced in uræmia, puerperal eclampsia, tetanus, acute yellow atrophy of the liver, various infectious diseases, gastric dilatation, hydrophobia, &c., produce general convulsions; and to the same category may belong the convulsions occurring in sunstroke.

Poisons introduced from without, as lead, strychnine, prussic acid, aconite, veratrum, urari, &c., may cause general convulsions; in many instances of poisoning the phenomenon is directly owing to the asphyxial condition produced by the retention of carbon dioxide in the blood.

In strychnine and tetanus poisoning the convulsions are of the tetanic type, and are probably entirely of spinal origin, and consciousness is intact.

The convulsions which are characteristic of epilepsy and hysteria will be described under the heading of these affections. The convulsions occurring in infancy require special mention.

CONVULSIONS, INFANTILE

Owing to the incomplete development of the higher nerve-centres at birth, and for some time after, the infant is liable to attacks of general convulsions, the lower centres being thus less under the control of the undeveloped upper ones.

Etiology and Pathology.—Convulsions occurring during the first few days of life are probably caused by hæmorrhages or injuries received during birth. Any organic brain-lesion, as tubercle, meningitis, infantile hemiplegia, hydrocephalus, &c., may be accompanied by convulsions in children, but such cases are not generally included under the term 'infantile convulsions.'

The main cause of infantile convulsions is rickets, but in the rickety infant there is often some exciting cause, as the irritation of teething, the presence of indigestible food in the intestines or stomach, round-worms, &c.; usually the convulsions in rickets are associated with laryngismus stridulus, carpo-pedal contractions, or tetany.

The tendency at the present time is to attach too little importance to teething troubles, as in former days too much was made of them. It is not a rare thing to find convulsions occur about the time that the crown of a tooth is making its way through the tissues above it, and these are met with sometimes in children in whom there can be no suspicion of

rickets, each successive tooth being heralded in with an attack.

Peripheral irritation in any region may excite an attack in infants that have not rickets, and the exanthemata and pneumonia are often ushered in with the convulsion which usually takes the place of the rigor in adults. Even the temporary congestion produced in a fit of coughing in pertussis is sufficient to determine an attack, whilst the asphyxial state in bronchitis and broncho-pneumonia often ends in convulsions from retained carbon dioxide.

The blood-condition in anæmia and after exhausting diarrhœa, as in the so-called hydrocephaloid state, may induce a general convulsion.

In many cases a history of inherited neurotic instability may be obtained in the mother or in the elder brothers and sisters of the child, and a percentage of the cases become epileptic later on, though the rule is that the tendency to convulsions diminishes as the upper centres reach their final development.

Symptoms.—These come on suddenly, and in the main correspond to the phenomena seen in typical epilepsy. The eyes get fixed or turned to one side, the pupils dilate, the face becomes white, the limbs and trunk become rigid from tonic spasm, and the respiration stops; the face gets purple, and then a series of general clonic spasms supervene during which foaming at the mouth is noticeable. The thumbs are turned in, and the fingers flexed upon them vigorously. During the attack, which generally lasts a few minutes, the sphincters may give way, and there is total unconsciousness. Recovery is followed by drowsiness, or by a series of similar attacks which pass into coma, and may in rare cases end in death. When the attacks succeed each other with great rapidity, the condition known as the status convulsivus is produced.

Often the attack is preceded by grinding of the teeth; muscular twitchings of the face and hands, and sometimes paresis of one side or strabismus may be noticed to follow the convulsion, if severe. Occasionally the spasms may be entirely tonic or confined to one side; but this latter is very rare, and it is easy to overlook spasms which may be much slighter on one side than on the other.

In rickets the attacks may continue at irregular intervals till the end of the second year.

Diagnosis.—The fact of the patient being an infant, and the nature of the attack being epileptoid, make the diagnosis generally easy, but it is always necessary to exclude organic brain-trouble. If the fits should be unilateral and always recurring on the same side, a cortical lesion should be suspected, and infantile hemiplegia will be the probable termination.

An attempt should always be made to diagnose the cause of the attack ; high fever when present is indicative of the onset of one of the exanthemata or of pneumonia. In meningitis other signs are sure to be present or to show themselves soon after the first seizure.

The beading of the ribs and enlargement of the epiphyses, with marked delay in the appearance of the teeth and the openness of the fontanelle, will demonstrate the presence of rickets, even during the progress of the convulsion.

Prognosis.—As regards the immediate result of the attack the prognosis is good, but it is always serious in the status convulsivus and in asphyxial conditions arising in whooping-cough and broncho-pneumonia and in chronic diarrhœa.

Where recurring unilateral convulsions are followed by hemiplegia, the case ceases to be strictly one of infantile convulsions, and the prognosis is grave as regards the future mental condition.

The remote prognosis is generally favourable, but it should be very guarded in those cases with a bad neurotic history ; such are prone to end finally in epilepsy, and the risk is greater if rickets be present. Idiocy follows, according to Gill, in about 3 per cent. of cases.

Treatment.—During the attack the less interference the better. All restricting garments should be removed, and the infant may be placed in the lap of the nurse or flat upon a bed. The warm bath is the usual routine, and it certainly does no harm ; and if it be no relief to the infant it always affords comfort to the bystanders and attendant to be doing something. A whiff of nitrite of amyl sometimes cuts the attack short, or prevents it in those cases where twitchings give warning of another attack. If the convulsion be severe, chloroform may be employed without hesitation ; and in prolonged attacks a dose of chloral (5 grs. for a child one year old) may be given with bromides by the rectum. When there is evidence of fever, cold or ice to the head is beneficial. Opium, though recommended by eminent authorities, has many serious drawbacks, and the young physician will be wise who discards it. The forefinger, thoroughly disinfected, may be passed into the mouth ; and if a tooth be presenting through the distended tissues of the gum, gentle scraping with the sharpened finger-nail till the crown is felt can do no harm. It is to be hoped that the gum-lancet will soon be only seen in museums.

After the attack the bowel should be cleared out by 1–3 grs. grey powder, or half as much calomel given in a little syrup of senna. It is most desirable to prevent every attack when possible, and the bromide-treatment should be continued for weeks or for months when threatenings are noticed.

The proper treatment for rickets or any intercurrent affection must be pressed.

The convulsions which occur in older children may be caused by intestinal troubles, often by gorges of unripe gooseberries and other fruit, and, in the opinion of Eustace Smith, to eye-strain and adenoids. These afford clear indications for treatment.

CORYZA, ACUTE

This is the name usually given to Acute Nasal Catarrh. It is, however, but a symptom of various conditions, as hay-fever, measles, influenza, &c. Acute nasal catarrh or acute rhinitis, commonly known as 'cold in the head,' is probably a microbic disease and contagious, occurring in epidemics. There is, however, a form which results from simple exposure to cold, and which is not infectious.

Symptoms.—These begin with sneezing, malaise, fever, pain over the frontal sinuses and brows, and obstruction of the nose, so that the respiration soon has to be entirely carried on through the mouth; smell and taste are blunted; and there is more or less sore-throat and slight deafness. The eye-ducts are blocked, like the nasal passages, by the swollen condition of the mucous membrane, and the tears overflow. Soon a free discharge of clear watery mucus flows from the nose, which, however, affords little or no relief, but causes incessant nose-blowings or trumpetings which cause much annoyance and discomfort. After a few days the discharge becomes opaque and finally more or less purulent, and the irritation creeps down to the larynx, trachea, or larger bronchi, producing hoarseness and cough; often herpes appears on the lips.

Inside a week the attack gradually subsides; but often there appears a very peculiar secretion constantly overlooked in descriptions of the affection, but often noticed when closely inquired after. This is the appearance of a bright yellow secretion resembling yolk of egg and entirely unlike pus, which is blown out of the antrum by using the handkerchief; it has a peculiar odour and its discharge is often associated with a dull pain about the fangs of the upper canine teeth.

Treatment.—If the patient can take to bed or remain in his room in an equable, but not too elevated, temperature, the symptoms may be cut short. A hot-air or Turkish bath is beneficial, but many cases are made worse by the ordinary warm bath. A full dose of morphia at night, combined with a strong diaphoretic, may give relief, but constant dosing by opiates in any form aggravates by drying up the secretion. A simple diaphoretic mixture containing $\frac{1}{16}$ gr. tartar emetic

and 1 dr. Mindererus spirit may be given every three or four hours. Some authorities believe in the abortive action of a large dose of quinine.

Of local remedies, as Ferrier's snuff, cocaine, menthol, eucalyptol, hamamelis, boric acid, &c., there is no end ; they too often aggravate the discomfort after affording transient relief. A 1 in 100 carbolic spray is perhaps the most uniformly unobjectionable local remedy.

CORYZA, CHRONIC

This name is applied to two different affections or forms of chronic nasal catarrh—viz. chronic *hypertrophic* and chronic *atrophic rhinitis*.

The *hypertrophic* affection is characterised by a thickening of the mucous membrane, especially over the inferior turbinated bones, with a varicose condition of the venous sinuses ; it is generally the result of repeated attacks of acute coryza, but may be induced by constant exposure to irritating fumes or dust. It is often associated in children with adenoids and enlarged tonsils, and in the adult with pharyngeal catarrh.

The obstruction in the nose causes continual discomfort, as all respiration must be carried on through the mouth, and there is much hawking or coughing, and sometimes sneezing.

Treatment.—The condition is an essentially chronic one ; but in mild cases considerable relief is afforded by the use of a fine spray of paroleine to which a little menthol has been added, the nostrils having been previously flushed out with a solution of boric acid and chloride or bicarbonate of soda, by means of the nasal douche or by sniffing it up from the hollow of the hand. In all severe cases the hypertrophied tissue must be removed in part at least by the cautery, caustics, écraseur, or snare.

Atrophic rhinitis is a disease of youth occurring most frequently in girls and young women, probably as a result of an earlier purulent rhinitis, caused by catarrhs, scarlatina, or adenoids. The atrophy involves both the mucous membrane and the turbinated bones ; the bridge of the nose is depressed and broadened, and the tip is turned up and the nostrils covered over with dry crusts. The chief characteristic is the almost constant presence of an indescribably fetid discharge or ozæna, so that the disease is often called ozæna, though this may be present in other nasal affections without atrophy, and at intervals in the atrophic form the nostrils may be dry. The sense of smell is lost, so that the patient becomes unconscious of the horrible stench. Both nostrils are always involved.

Treatment.—The disease is practically incurable, but much may be done by constant douching with antiseptic lotions, a long list of which may be found in the author's 'Dictionary of Treatment' (4th ed.), pp. 653–656.

COUGH

This is but a symptom, and though usually a symptom of disease of the air-passages, lungs, or pleura, it may be the result of several affections outside the chest.

Etiology and Pathology.—Cough is generally an involuntary reflex act commencing in the closure of the false vocal cords after a full inspiration; the expiratory muscles are then brought suddenly into action, and the air in the chest is compressed and forced out through the larynx, carrying with it the secretion or foreign body causing the irritation.

The nerves which convey impressions compelling the action of the cough-mechanism are chiefly the branches of the vagus distributed to the mucous membrane of the larynx, trachea, and bronchi; but the naso-pharynx, with its host of ailments, each of which may cause nerve-irritation, plays a considerable part in the production of this symptom. Thus nasal polypi, rhinitis, ulcerations, coryza, crusts, and various bone or cartilage lesions involving the mucous membrane, adenoids, enlarged tonsils, the various forms of pharyngitis, elongation of the uvula, diphtheritic exudation, and even enlargement of the so-called lingual tonsil at the base of the tongue, may all produce coughing.

Ear-cough is not rare. It is caused by irritation of the nerve-endings in the meatus, and can be produced in most persons by tickling the floor of the auditory passage; wax, foreign bodies, or the irritation produced by the discharge in otitis may cause it.

Stomach-cough is an occasional symptom of dyspepsia or gastric catarrh; liver-cough and spleen-cough are recognised: they are probably induced through pressure on, or irritation of, the diaphragm. Uterine or ovarian irritation is also credited with the power of producing cough.

Mediastinal tumours, as aneurisms, enlarged glands, pericardial effusion, abscesses, or vertebral disease, or even cardiac hypertrophy, induce coughing. Valvular diseases and failing compensation produce cough generally by causing some bronchial congestion. Nervous coughing is often an acquired habit, and hysteria is frequently the cause of a formidable persistent cough. As a rule, the so-called transferred or reflex cough, or cough induced by conditions other than bronchial catarrh, is a dry, spasmodic, persistent hacking or barking.

The barking cough of puberty, first described by Sir Andrew Clark, occurs in boys who are often the offspring of neurotic parents.

From the above list it becomes obvious that few greater mistakes can be made than that of regarding cough as a disease or as a symptom confined to pulmonary disease, and in every case the diagnosis of the cause should be carefully worked out before rational treatment can be decided upon.

Diagnosis.—The cough in *pertussis*, with its series of prolonged expiratory efforts, followed by the whoop or deep-crowing inspiration, cannot be mistaken for any other affection.

Laryngeal cough, caused by ulcerative, inflammatory, or diphtheritic states, is accompanied by huskiness or hoarseness; it is often described as croupy. Where the membrane is inflamed and dry, the character is brassy, ringing, or clanging, and this is the main feature of the cough produced by aneurismal or other tumours pressing upon the recurrent laryngeal nerve. When both vocal cords are paralysed, the imperfect cough is accompanied by the peculiar bovine character of a prolonged grunt or wheeze. Stridor and dyspnœa, accompanied by a paroxysmal clanging cough, is highly characteristic of aneurismal or other tumour-pressure, and more or less aphonia may be present.

Bronchial inflammation produces a cough which, in the first stage, is dry, short, and painful; afterwards it becomes moist or loose and painless, and the secretion is characteristic.

Pneumonic and *pleuritic* cough resembles that of the first stage of bronchitis, but it is more frequent and restrained by the characteristic intense pain in the side; usually the two forms cannot be differentiated, as in pneumonia there is generally some pleuritis present, though both diseases occasionally occur without any cough at all.

Phthisis has in its early stage a dry, incessant, hacking, and often painful short cough which may be almost diagnostic; in the later stage it is loose, and resembles that of chronic bronchitis.

Bronchiectasis is diagnosed often by the cough alone: it is paroxysmal with long intervals of rest; the act brings up large quantities of often fetid pus which have been accumulating during the period of rest, and a change in the position of the patient may at any time bring on a paroxysm, as in the act of stooping.

Treatment.—This will entirely depend upon the cause, and under each heading the appropriate remedies will be referred to; but a word may here be said as to the general principle which should guide the physician in his decision as to whether this symptom, as a symptom, should receive any drug-treatment or not. Where a *dry*, painful, and severe cough is persistent, it should, if possible, be relieved by sedatives which diminish

the activity of the respiratory centre, as the increased pressure in the air-cells is injurious, and the patient's rest is seriously interfered with. Opiates alone or combined with bromides, or in the case of children small doses of chloral, may be given. But when the cough, though persistent, is always accompanied by secretion, and especially by copious secretion, sedatives are always injurious. The only remedies in these cases admissible are drugs which act upon the diseased membrane and alter the character of the secretion—viz. true expectorants. The common mistake of confusing sedative expectorants with narcotics has been emphasised under 'Bronchitis.'

CRAMP (WRITER'S)

This is one of the group now recognised as occupation-neuroses, in which painful spasm sets in in the muscles employed continuously in any special act. Thus there is a telegraphist's, sempstress's, cigar-roller's, pianoforte- and violin-player's cramp.

Etiology. — Writer's cramp, or Scrivener's palsy, chiefly affects males from twenty to forty years of age. There is sometimes an hereditary family history, and often a history of mental worry or of over-work as regards writing; occasionally the affection comes on after some local injury. But according to Gowers faulty methods of moving the pen along the paper in the act of writing are the chief causes, as when the little finger or the wrist is used as a fixed basis of support for the hand. In those who write from the middle of the forearm or from the upper arm and the shoulder the affection is seldom, if ever, seen; thus it is almost unknown in stenographers, and is very frequently seen in law clerks and other copyists.

Symptoms.—The spasm or cramp generally comes on slowly, and may affect the thumb or first and second, or in rare cases all the fingers of the hand; the pen is held too firmly, the writing becomes changed and irregular, and the letters tend to become smaller with an increasingly thick downstroke, and finally writing becomes an impossibility. The spasm is generally tonic; but occasionally a clonic spasm occurs which causes the pen to be jerked about. The difficulty is usually exaggerated by mental excitement and by the concentration of the attention upon the formation of the letters. Pain is nearly always present, and often this takes the form of a dull, tired feeling in the fingers, but not infrequently it is severe in the wrist till the hand is rested; it may be present without spasm, and may take on a neuralgic character. Tremor and paresis may be present, but they are nearly always associated with some spasm, so that pure tremulous or paralytic forms are rare.

Other delicate muscular movements of the hands may not be appreciably affected, the cramp only showing itself when an attempt is made at writing. The electrical reactions are normal, but become slightly increased, and finally, in long-standing cases, diminished, the interossei most frequently showing marked sluggishness in their response both to the faradic and galvanic current.

Pathology.—Various theories are held regarding the pathogeny of the affection, the most probable one being that which attributes it to an altered state of the nutrition in the cortical centres presiding over the movements. There is diminution of the resistance to impulses passing between the various commissural paths connecting these centres, and a condition of irritable weakness results. Another theory is that the voluntary impulses travel downwards along other motor nerves, bringing sympathetic movements into action which impede the intended efforts. Poore regarded the pathology of the condition simply as that of chronic fatigue of the muscles employed in pen-prehension, notably the first and second dorsal interossei and the thenar muscles; but he regarded the pathology of other occupation or craft palsies, as in hammermen and nailmakers where the movements are never automatic, to be cerebral fatigue, for in these cases the cerebral effort is out of proportion to the mechanical or muscular work.

Diagnosis.—This is never difficult, since writer's cramp can hardly be mistaken for any other affection; minor forms of hemiplegia, chorea, and progressive muscular atrophy may be mistaken for it when the primary lesions are overlooked, but these are easily, with care, traced to their respective categories. Many persons fancy they are the subjects of the affection after experiencing fatigue caused by a spell of unusually severe writing or copying; a careful inspection of such individuals, when asked to write a few dozens of words, will speedily demonstrate their freedom from spasm, tremor, and pain.

Prognosis.—This is only serious in those who have to earn their livelihood by writing, especially in the lawyer's clerk, whose chief work consists in copying. If the affection has been of long standing, it will probably prove incurable, especially in those whose health has already suffered from over-work or depressing mental anxieties. The prognosis is good if the affection be taken in time, and if the patient can take a long rest, or if he can be induced to persevere in learning to write with his left hand, though in some cases this also may become the seat of spasm.

Treatment.—Rest is of first importance, and the duration of it must depend upon the severity and duration of the patient's symptoms. A month or two of absolute rest from all

writing at the beginning may stave off the affection, whilst it will take as many years at a later stage to make any impression upon the disease. The Gowers treatment of making the patient write a free hand from the shoulder should always be insisted upon. A quill is preferable to a steel pen; the various devices of penholders, bracelets, and the like are, as a rule, valueless, as they entail in the long run more real labour.

The patient should learn to write with his left hand, or, if the act of writing is impossible, he must either change his occupation or take to typewriting.

Massage and electricity may be tried—the latter in the form of the continuous current combined with rhythmical movements of the fatigued muscles. Gymnastic exercises which bring the muscles into actions with a different rhythm from that of writing are of use. Nervine tonics are indicated in some cases. The other craft-neuroses, spasms or palsies as they are called, must be treated on similar principles.

CRAMP (SIMPLE)

This is the name applied to a tonic spasm accompanied by severe pain in the muscles, chiefly in those of the calf or thigh at night, and often seriously interfering with rest and sleep. The etiology of the condition is obscure; it may be of reflex origin, or due to over-fatigue of the muscles, or may arise from some toxic condition of the blood. The best method of cutting short the painful rigidity is by a strong effort of the will to bring into action the opposing extensors. Sometimes a thick cord applied above the contracted muscles and drawn tightly like a tourniquet may stop the tetanic spasm.

Swimmer's cramp is a peculiar and serious condition about which little seems to be known. The writer has experienced it in his own person upon two occasions when a considerable distance from the shore. The popular idea is that it is a tetanic spasm in the muscles of the leg, arm, or other region akin to the cramp just mentioned. This is a mistake; such spasms are common in swimming, but the expert swimmer, by giving the rigid member a rest whilst he turns on his back and floats or uses the opposite side of his body only, soon gets relieved, and he may continue his exercise in deep water with safety.

In the so-called cramp which so often leads to drowning the condition is widely different, and appears to be, in the writer's opinion, a sudden suspension of the will-power of the swimmer over every muscle of the body; and few escape to narrate their strange experience. The only phenomenon or condition akin to it is that which sometimes seizes upon a public speaker and leaves him absolutely powerless to utter a thought or word, though consciousness is active and all sensation of pure nervousness may have vanished.

CRAW-CRAW

This is a term from the West Coast of Africa which is applied to obstinate skin-diseases associated with much itching. By some authorities, including Manson, it was supposed to be the result of a filarious blood-worm ; it is now held by many to be but a neglected form of scabies, and this seems to be the probable cause of the variety common in Uganda, which is cured by ordinary itch-remedies like sulphur.

CRETINISM

This may be defined as congenital myxœdema—a condition originating in the absence of the thyroid secretion, characterised by arrest or retardation of the bodily and mental development, producing a remarkable kind of deformity.

Etiology and Pathology.—It is either *endemic*, as in the mountainous regions of Switzerland and Northern Italy, or *sporadic*, cropping up in various districts scattered over the globe. In both forms the thyroid gland is either absent or atrophied, or it is the seat of goître. The cause which leads to the wasting or absence of the gland is unknown ; it is conjectured to be a microbe in the drinking-water.

Symptoms.—The disease appears about the sixth to the ninth month of the infant's life, when it is noticed that there is some mental or physical 'backwardness' : the child has ceased to grow like other infants, and it is dull and less observant. The tongue seems too large for the mouth, which remains generally more or less open, giving an absence of expression to the face which even at this early stage can scarcely be mistaken ; the skin is dry, the temperature subnormal, and the hair scanty. Before the middle of the second year the typical condition has developed ; the child measures much less in length than the normal, though his face looks large and somewhat coarse, his nose being broad and his eyelids puffy-looking. The fontanelles remain open, the teeth are delayed and decay early, the hands are broad and the fingers short, and the mental deficiency is so marked that idiocy is suspected. Later on, in less severe cases, the child learns to say a few words and gradually becomes able to combine them. All degrees of mental backwardness may be seen in myxœdematous children as they grow older. The abdomen becomes prominent, umbilical protrusion is always present, and lordosis occurs ; large pads of fatty tissue appear above the clavicles and in front of the armpits ; the puffiness of the face increases and spreads to the feet and hands ; the skin and the hair get drier and harsher. A cretin at twelve

years may be mistaken for a child of three, his movements are slow and waddling, and all cretins bear a close resemblance to each other. The stunted stature and retarded mental development are more striking as the cretin reaches to adult age, when he may measure not more than three feet, with a disproportionately large head of the brachycephalic type and thick-set short limbs which are always cold, and dry skin, and hairless undeveloped genital organs. A similar arrest in the sexual organs is noticeable in female cases, where the breasts do not develop and menstruation may never appear. As a rule, in endemic regions the cretins possess a degenerate thyroid gland, which is obviously enlarged by some goitrous change ; but in the sporadic cretins the gland is so wasted that often its presence cannot be detected.

When the disease first appears in later childhood (which it sometimes does after an infectious fever) the condition has been termed 'juvenile myxœdema,' and the symptoms are modified only by the fact that the development has been normal up to the period of the commencing wasting of the thyroid gland. The cretin, if not placed under treatment, may continue to spin out his listless life to old age, his faculties to the end remaining in their child-like undeveloped state.

Diagnosis. — The condition once seen can scarcely be mistaken. It may be taken as the type of *Infantilism.*

The *Mongolian type of idiocy* may simulate cretinism ; but, though the stature is somewhat stunted, it is never so markedly so as in cretinism, and the Chinese physiognomy, with the obliquity of the eyes and the absence of the apathetic listlessness of the cretin, and their marked imitative powers, are characteristic.

Fœtal cretinism, so-called, is a condition formerly supposed to be allied to rickets which may be mistaken for the disease under consideration. But the deformity exists only in those bones which should be ossified from cartilage before birth occurs ; the bones developed from membrane and those from cartilage late in intra-uterine life are normal. The extreme shortness of the limbs at birth, and later on the normal state of the hair, skin, genitals, and mental faculties distinguish this dwarf condition, which is also known as Achondroplasia or Chondrodystrophia fœtalis. (See also under 'Infantilism.')

Prognosis.—This has been dealt with in the description of the disease ; but it may be said to be entirely dependent upon treatment if the condition has been recognised early, and almost marvellous results are obtainable.

Treatment.—This is the same as for myxœdema, and consists in the continuous administration of thyroid gland or some preparation made from it, which must be administered all through life. Dosage is important, and, as a rule, the doses

advised are much too large. For an infant eight to twelve months old, 1 min. daily of the B.P. liquor is enough: this corresponds to $\frac{1}{100}$ of a sheep's thyroid gland. If improvement does not soon become obvious and progressive, double this amount may be given. After the first years of treatment the dosage may be but once or twice weekly in larger amounts—5 grs. of the dried gland. Every departure from the normal is corrected by the thyroid as long as its administration is attended to.

In cretins who first come under notice and treatment in childhood, say at the age of eight to ten years, a dose of 5 mins. daily, increased if necessary to 10 mins., may be given. The stature, which had been permanently fixed, in these cases begins to increase, and a growth of six to eight inches inside one year is not unusual.

In cretins up to twenty years of age, similar results may be expected; but there is a great tendency for the legs to become bowed as in rickets. The mental improvement keeps pace with the physical, but a certain amount of 'backwardness' may be expected to remain.

Older cretins generally grow but little in stature after the commencement of the treatment, though their mental state brightens considerably, even when no increase of height is noticeable.

In all cases, whether infantile or adult, the cretin slowly and steadily returns to the myxœdematous condition upon the suspension of the thyroid feeding, though of course his stature does not diminish. The great object should be to determine the minimum dose necessary to keep him up to the normal standard; this may be accomplished by careful weighings and counting the pulse. A fall in body-weight and an increase in the pulse-rate, with elevation of temperature, indicate that the safe constant dosage has been exceeded. In many cases 5 grs. of the dried gland twice weekly will be found sufficient. There is no advantage in administering the colloid or other supposed active principles separated from the thyroid-gland substance.

CROUP

This term is applied to three distinct affections which should not be confounded with each other: (1) *Membranous laryngitis*, formerly known as true croup, and now by common consent regarded as diphtheria; (2) *laryngismus stridulus* or child-crowing, a state of simple spasm of the glottis often seen in rickets; (3) the affection known as *false* or *spurious croup*, *inflammatory croup*, *laryngitis stridulosa*, or *spasmodic croup*, and with this latter the present article alone deals.

Etiology and Pathology.—False or spasmodic croup is a disease of childhood occurring between the ages of eighteen

months and four years. The pathogeny of the affection is a slight laryngeal catarrh which induces spasm of the muscles of the larynx. It is possible that spasm may occur without laryngitis; but, undoubtedly, in the great bulk of these cases of false croup an element of laryngeal inflammation is always present. Very slight tumefaction of the mucous membrane at the upper part of the larynx in young children makes an appreciable diminution in the size of the opening.

Symptoms.—Its onset is always sudden and alarming. The child, who has been put to bed in its usual good health, awakes during the night with difficulty of breathing and a characteristic hoarse croupy cough; the same husky character is noticeable in the voice or alarmed cry of the child. A crowing sound may accompany inspiration. Lividity or cyanosis appears in proportion to the dyspnœa.

The attack passes off as abruptly as it came, and in less than an hour the formidable symptoms of suffocation, which may have lasted several minutes, have completely subsided, and the child falls into sound natural sleep. From this in a few hours it may awake to pass through a somewhat longer attack, which may be repeated again upon following nights. Generally speaking, after an attack, recurrence is to be expected to follow any undue exposure or catarrh till the child gets into his fifth year. Often slight laryngeal symptoms, as hoarseness, may remain for a day or two after the night attack. Slight feverishness is often present.

Diagnosis.—The important point is the exclusion of the far more serious membranous or *true diphtheritic croup*, and this may be difficult where no exudation is visible about the fauces. The fact of the perfectly healthy condition of the child a few hours before the attack is of vital importance, as in diphtheritic croup there is sure to be a history of malaise and hoarseness of the voice prior to the patient going to sleep; and, moreover, the true croup, when sufficiently advanced to cause laryngeal spasm, does not rapidly pass off, and the voice is not merely hoarse, but whispering: in other words, there is more or less aphonia. In nearly every case where the physician is called to witness a sudden attack of alarming laryngeal obstruction occurring in the night-time in a child who has been known to be quite well a few hours before, he may be sure he is dealing with false croup, which is always *apparently* more serious than the early stages of the true variety.

In *laryngismus stridulus* the attack may be alarmingly sudden also, but there is no cough or hoarseness; the respiration is totally arrested for a few seconds, after which the spasm relaxes and the air enters the trachea with a crowing sound. The attacks may occur very frequently during the daytime, and be associated with carpo-pedal spasms, or even a general convulsion; and signs of rickets are very often present.

Œdematous laryngitis is recognised by the sudden total loss of voice, dysphagia, urgent dyspnœa, and hissing inspiration which follows scalds of the larynx after inhaling steam or swallowing boiling water or corrosive liquids.

Prognosis.—This is always favourable; death probably never occurs in pure spasmodic croup.

Treatment.—The attacks yield rapidly to emesis. Ipecacuanha or tartar emetic should be given immediately and pushed till free vomiting occurs; in their absence any emetic may be given or tickling of the fauces may be tried. The symptoms also yield rapidly to a brief inhalation of chloroform, and the hot bath aids the emetic. Nitrite of amyl and nitroglycerin are also prompt in their action.

CYANOSIS

Is the term used to indicate the blue, livid, or purple condition of the surface of the body caused by imperfect aëration of the blood. It is such a constant phenomenon in congenital heart-disease that the word is used as a synonym for this affection, which is also known as *Morbus cæruleus*. In ordinary conditions of embarrassed respiration cyanosis is first noticed in the finger-tips, lips, and lobules of the ears. Where the vena cava superior is obstructed, cyanosis may be confined to the upper half of the body, and it may be localised in any region owing to pressure on its venous trunk.

A typical cyanosis occurs in some persons from the administration of antifebrin, and it is marked in poisoning by drugs which depress the respiratory centre or interfere with the elimination of carbon dioxide. All diseased conditions which impede the admission of air to the air-passages or alveoli and all diseases which interfere with the pulmonary circulation cause cyanosis; thus it is seen in tumours pressing upon the larynx, trachea, and bronchi, and in inflammatory states of these passages, when it becomes with dyspnœa the obvious symptom of suffocation. In diseases like pleuritis with effusion, pneumonia, œdema, or collapse of the lung, miliary tuberculosis, large tumours, or pericardial effusions, emphysema, and great abdominal distension, the air-cells are compressed or obliterated and the aërating surface so diminished that cyanosis results.

Valvular lesions, when associated with failing compensation, cause cyanosis by the failure of the engorged right ventricle to drive the blood through the pulmonary capillaries.

G. A. Gibson claims recently to have demonstrated a hitherto undescribed disease or form of cyanosis produced by the presence of the Bacillus coli in the blood. This leads to the hæmatogenous formation of nitrites, causing methæmoglobin and sulphæmoglobin, which produce cyanosis. The term *microbic cyanosis* is applied to the condition.

DELIRIUM

This is a state of deranged or perverted consciousness arising from disturbances in the grey matter of the convolutions, characterised by incoherence of thought and speech and often associated with delusions, illusions, or hallucinations. Gowers states that the condition of delirium is essentially the same as that which constitutes insanity; but the term should be confined to the acute mental derangement which is secondary to organic brain-disease, pyrexia, toxæmia, &c.

Three common forms of delirium are usually recognised:

1. *Delirium tremens*, which will be separately described.

2. *Active, wild*, or *raving delirium*, which may closely resemble *D. tremens*, but has not the same causation. In this state, frequently seen in the early stages of severe fevers, meningitis, &c., the patient is often violent and requires restraint, and puts forth greater muscular power than he could be believed to possess; suicidal tendencies may be present; he raves loudly and appears to act upon the delusions or false ideas which occupy his mind or upon the hallucinations which arise in his brain, regardless of any sensory impressions produced by his environment, to which indeed he may seem oblivious.

3. *Low* or *muttering delirium*, seen in the late stages of continued fevers, pneumonia, &c. The patient lies in this condition without marked bodily restlessness, rambling about delusions which run through his disordered brain or about hallucinations produced without sensory impressions; or he may be continually suffering from illusions which are produced by perverted sensory impressions: such illusions are frequently seen in typhus. The condition is often accompanied by carphologia, subsultus, or floccitation, and usually more or less stupor. Uræmia and poisoning by various drugs, hysteria, epilepsy, and many other conditions, especially those following inanition, produce more or less delirium.

DELIRIUM, ACUTE

This term is applied to an acute mental disorder, generally ending fatally, characterised by feverishness, active delirium, intense prostration, and rapid wasting.

The disease is known by various names, as Bell's Mania, Typhomania, Acute Delirious Mania, Brain Fever, Phrenitis, &c.

Etiology and Pathology.—The onset is rapid, but a family history of insanity and some preceding debility, depression, or insomnia are common. Nothing is known of the pathology; it is regarded as either the result of a microbic infection or a toxæmia from some poison manufactured in the bowel.

Symptoms.—Delirium shows itself by evidences of hallucinations, and all sorts of disorders of consciousness rapidly appear; the patient becomes most restless and violent, and soon passes into a state of furious mania, with incessant ravings or chatterings. Sleep is always absent; the patient refuses food, and resents violently all attempts at forced feeding; and, as a result of his perpetual excitement, by the end of the second day he may be in a condition of profound prostration. The temperature may reach 105° at this stage, but it is generally lower; the pulse is quick and weak, the tongue dry and brown; there is marked wasting. Gradually restlessness, twitchings, and low muttering delirium takes the place of the former maniacal excitement; the patient becomes apathetic; stupor appears, which passes into coma, or he dies from failure of his cardiac muscle within a week of the onset of his frenzy. Some cases may linger longer after the violence of the early excitement has passed off.

Diagnosis.—The rapidity of the onset, the intensity of the delirium, and the early tendency to grave prostration, with marked fever, distinguish the disease from *ordinary acute mania.*

Typhoid fever sometimes, but rarely, is ushered in with wild delirium, especially in cases where alcoholic excess has been habitual; headache is a constant feature in such cases, but it stops before the delirium commences; epistaxis and other signs of the disease are generally present.

Organic brain-disease and *meningitis* are excluded by the history of the onset, the absence of convulsions, of eye-paralysis or other focalising symptoms, and of optic neuritis.

Delirium tremens is easily recognised by the history alone, and this is nearly always available; but there are some cases which begin with the wildest delirium and distressing hallucinations and closely resemble acute delirious mania; tremor is, however, always prominent, and the temperature is not markedly elevated; though the excitement may approach that of acute delirious mania in its intensity for a short period, there is never the profound and rapid prostration which marks the graver malady.

Prognosis.—This is always grave; nearly all the cases die. It is possible that some of the reported recoveries were cases of ordinary acute mania; patients do recover from the acute delirium sometimes, but total dementia is liable to supervene.

Treatment.—Early removal to a suitable institution, provided with a skilled nursing staff and isolation-rooms capable of excluding light, and appliances for forced feeding, &c., is necessary. From the earliest symptoms the feeding should be attended to, and the œsophageal tube used if necessary; absolute quiet and simple hypnotics, as trional,

sulphonal, and paraldehyde, must be used to induce sleep. When the frenzied stage is over, liberal stimulation is imperative to prevent a fatal collapse.

DELIRIUM TREMENS

This is the name given to a condition of the nervous system supervening usually upon prolonged alcoholic excess.

Etiology and Pathology.—Chronic alcoholism is the factor in nearly all cases. It may, however, follow a single prolonged debauch or acute bout of drinking. The typical factor producing the disease in the great majority of cases is one unusually severe bout of drinking, in an habitual drunkard. The attack may be precipitated in such an individual by any accident, injury, or disease which may overtake him during his course of steady drinking, under which circumstances it is often spoken of as 'traumatic delirium'—an unfortunate term liable to be confused with the delirium associated with sepsis and other conditions.

The pathology of delirium tremens is best explained from the standpoint of a chronic toxæmia induced by alcohol, which produces an irritable or unstable condition of the cortex and deeper nerve-centres, in marked contrast to the paralysis resulting from acute alcoholic poisoning. The changes found after death consist of minor forms of congestion of the cerebro-spinal system, sometimes confined to the cerebral pia mater, plus the gross changes found in the liver, kidneys, and heart, the result of long-continued abuse of the narcotic poison. Very generally congestion of the bases of both lungs is noticeable.

Symptoms.— The onset may be sudden, but there are generally premonitory symptoms felt and realised, often by those who have had frequent attacks ; it is, however, difficult to unravel the tangled web of symptoms which precede the attack, because these are but an accentuation of what may be noticed in every inebriate. Nausea is generally present in an aggravated form, so that often when the attack comes on the victim has been eating little or nothing for some days previously. The loathing often extends to alcohol ; hence the popular delusion that the delirium is caused by the sudden stopping of the stimulant. Statistics, and the experience of gaol-surgeons, are opposed to this idea.

Increased restlessness and insomnia, or broken, disturbed sleep, from which the patient starts up somewhat alarmed, with the delusion that he has heard voices calling to him, are amongst the most frequent premonitory symptoms. His distressing dreams pass imperceptibly into painful waking hallucinations and illusions. He sees various loathsome animals or small demons creeping about his bed, he hears

voices which instil terror, and he often mistakes his attendant for some dread foe, or for the Arch-enemy of mankind, and under this illusion he may make a sudden and fierce onslaught upon him, though the next moment he may embrace him as his friend. More frequently his terror impels him to escape from his environment, and he struggles to get out of bed and through windows and doors, and makes a dash in his nude condition for the liberty of the street or fields.

Chattering, incoherently talking, or loudly shouting, nevertheless he may be suddenly pulled up to transient sanity by some question or command of his attendant, but is soon off again, struggling with his delusions, hallucinations, or illusions. He never keeps upon one track of false ideas for long, the restless state of his brain keeping time with the perpetually changing condition of his physical restlessness.

Tremor is present in every case. At the beginning of the attack, or immediately preceding it, this feature seems to be but an exaggeration of the tremor characteristic of chronic alcoholism; but it speedily becomes general, and under the terror of the hallucinations all the muscles of his body are upon the tremble. Some of the many popular names of the disease are based upon this symptom—viz. the 'shakes' or the 'jigs.' Another popular name, the 'blue devils,' has arisen from the frequency with which the patient fancies that his phantom-illusions are of a blue colour, and the invariably painful or dread-producing character of the mental disorder has produced such names often given to the malady as the 'terrors,' the 'horrors,' &c.

The pulse is quick, compressible, and weak; there is always more or less perspiration, the tongue is always moist and flabby and covered with a thick uniform layer of white fur, and the breath is offensive and characteristic throughout the attack. Fever, sometimes absent, is usually present in severe cases, but it is never high, unless the delirium is very wild, or some complication like pneumonia sets in; and this should always be watched for. Insomnia is invariably a most prominent feature; and when sleep occurs, as a rule the patient awakes greatly improved or completely rational and sane, though tremor and weakness remain for some days. It is probable that sleep in all cases of severity, where it does occur, would set in, regardless of the enormous doses of morphia and other hypnotics usually administered, and this consideration has a most important bearing upon treatment, as the natural tendency of the disease is to terminate in spontaneous sleep, in the majority of cases, about the third or fourth day. Where sleep does not set in, the patient gradually sinks into a typhoid state, with low muttering delirium, which may pass into coma and death; or he may sink from heart-failure or pneumonia. In those who have

had repeated attacks, a severe general convulsion may wind up the attack, though recovery does occasionally follow when convulsions have occurred. Sometimes the convulsion heralds in the attack, and then it is a less unfavourable sign.

Recovery may, as regards the mental state, be incomplete, and the writer can recall several cases where the insane victims had to be passed into asylums.

Diagnosis. — The history and characteristic symptoms leave little room for difficulty in most cases. Somewhat similar delirium is witnessed in morphio-maniacs on the suppression of the narcotic, but this is at once relieved by a full dose of the drug, tremor is less marked, the hallucinations do not inspire such terror, and the attack is much shorter.

Insanity, especially in its maniacal form, may be mistaken for delirium tremens, but the special feature of the terror or horror so characteristic of the alcoholic delusions, or hallucinations, and the marked tremors, are absent.

Acute fevers and *pneumonia*, especially of the apex, may produce marked delirium, which is sometimes mistaken for the alcoholic affection; but unless these occur in an alcoholic patient, close observation of the patient's demeanour and the high temperature soon settle the diagnosis. The only point of importance worth emphasising is the danger of overlooking serious diseases, like pneumonia and meningitis, or internal injuries in subjects who are suffering from the true alcoholic affection.

General paralysis of the insane may lead to confusion if tremors be well marked, but the history of the exaltation, even though the patient should be also addicted to occasional alcoholism, clears up the case easily.

Prognosis.—First attacks generally end in recovery; the prognosis becomes more serious in each recurrence, and there is a popular notion that special danger attends upon a third attack.

The presence of any chronic disease, as kidney-affections, liver-cirrhosis, cardiac degenerations, or failing compensation, deepens the prognosis. Intercurrent affections and surgical injuries cause gravity in proportion to their severity and nature; pneumonia, erysipelas following scalp-wounds, and enteric fever are liable to be fatal. The supervention of hyperpyrexia, convulsions, especially upon the third or fourth day, and the total inability to retain or digest food, are symptoms of grave omen in all cases.

Treatment.—The patient should be immediately placed under the care of two skilled and experienced nurses, who are not to relax their oversight for a single moment. A quiet, darkened, and if possible a padded room, should be selected, with good fastenings to door and windows; these latter should be protected. Restraint must in almost every

case be resorted to, and a tactful nurse is able to keep a patient on his back in bed for hours, whilst a less skilful attendant will be engaged in continual struggles with him all over the sick-chamber. When forcible restraint is absolutely necessary, a sheet may be so tucked in at the sides as to hamper all violent movements. The old-fashioned strait-jacket should never, if possible, be employed, but in cases of extraordinary difficulty it is less objectionable than the sheer force which otherwise must be resorted to.

Feeding is of vital importance, and concentrated liquid nourishment must be forced upon the patient at short intervals. In the presence of collapse or cardiac or pulmonary complications, alcohol must be given freely with the food.

Owing to the fact that rapid recovery follows natural sleep, the physician is much tempted to administer large doses of narcotics or hypnotics; but these must be used with great discretion. The cast-iron rule of withholding hypnotics in every case, or of pushing them as a matter of routine from the commencement in every case, is to be strongly condemned. They are only indicated about the third day, when the perpetual drain upon the patient's strength begins to tell—a period when natural sleep may reasonably be expected to be within measurable distance—and by precipitating this desirable event by some hours life may be saved.

As a matter of routine, however, bromides may be given from the start; they can do no harm in moderate doses with 1-oz. doses of Mindererus spirit.

As to the choice of the hypnotic there is great diversity of opinion, but the writer does not hesitate to condemn chloral, which he believes is answerable for many of the sudden deaths recorded in delirium tremens. The safest drug is paraldehyde, in doses of 100 mins.; hyoscine hypodermically in quantities of $\frac{1}{100}$ gr. often acts speedily. Trional and sulphonal may be tried. But frequently all these hypnotics fail; then morphia must be resorted to, and the best procedure is a full dose hypodermically, combined with strychnine. (For details regarding drug-treatment, see the author's 'Dictionary of Treatment,' 4th ed. pp. 191–196.)

DEMENTIA

This condition in its numerous forms may be defined broadly as acquired mental enfeeblement. When it appears at the commencement of life the congenital form of mental weakness is called idiocy, a milder acquired form being usually termed imbecility. Late in life dementia is often met with as the outcome of the degenerative changes characteristic of senility. In *amentia*, as ordinarily defined, there

are no degenerative lesions found in the brain, which simply shows arrested development.

The great majority of the cases of dementia are secondary to the various forms of insanity, as mania, melancholia, &c., which come on after puberty, under which circumstances the disease is practically hopeless. Thus, a large proportion of the chronics which fill the lunatic asylums are examples of dementia which have passed through the acute phases of lunacy or have become weakened in mind after attacks of apoplexy, epilepsy, alcoholism, syphilis, &c. Where this condition supervenes as a sequel to insanity it is termed *secondary* dementia. In the early stage of the affection, following mild attacks of mania, recovery may follow the cessation of confinement and a total change of environment.

Primary dementia occurs in young subjects, sometimes in an *acute* form, especially in girls under twenty, owing to depressing causes, over-work, under-feeding, exhausting fevers, &c.

There is rapid and complete loss of all mental power, ending in a state of mental stupor, in which the intellectual and moral faculties are in complete abeyance.

These apparently hopeless cases often end in complete recovery under forced feeding, massage, and change of environment. Under the confusing name of *Dementia præcox* precocious or premature dementia is described by Kraepelin, but he includes in it the catatonic and paranoid forms, which may come on late in life. Easterbrook considers that the term *chronic mental confusion* is more appropriate, since in a considerable proportion of the cases there is but an apparent tendency towards dementia, which latter may never supervene.

Chronic primary dementia commences slowly, generally with loss of memory, and in the majority of cases follows apoplexy, epilepsy, syphilis, cerebral tumour, or chronic alcoholism, and often as a phase of the general changes met with in senility. The prospects of improvement lessen in proportion to the slowness or insidiousness with which the symptoms have crept on, and to the nature of the brain-lesion which they have followed. Such cases as are caused by alcohol and syphilis may improve under treatment directed to these ailments, and epileptic cases may improve if the convulsive seizures can be checked.

Paralytic dementia is another name for general paralysis of the insane.

DENGUE

This is an acute specific epidemic disease occurring in the tropics or in hot climates, characterised by a fever of rapid onset and short duration, with severe joint and muscular pains, accompanied by skin-eruptions.

It is known also as Breakbone Fever and by a variety of local names, and by the synonym of Dandy Fever, which it has received from the peculiarly stilted affected gait which it causes.

Etiology and Pathology.—Numerous epidemics of dengue have been recorded since the first description of it was published in 1779, during a great invasion by the disease of a large portion of the tropical world. It has appeared as far north as Philadelphia and the south of Spain in hot weather, and it has become endemic in the West Indies, Egypt, and Cyprus; but its marked tendency is to burst out after long intervals of quiescence in a pandemic form over vast regions, including the whole of India and Burmah, and even China, spreading rapidly along the lines of human intercourse or trade-routes. It surpasses influenza in the power which it possesses of seizing at once upon whole communities, only a very few escaping by reason of some natural immunity.

Its highly infectious nature and the short period of incubation (which is only one to three days) account for the rapidity with which it assumes the pandemic form; but this lightning-like rapidity of its spread has induced many to believe that the disease is not truly infectious.

The pathology of dengue, however, cannot be seriously doubted. It belongs to the exanthematous group of fevers, and is caused by a microbe which has yet to be isolated. In some respects it resembles scarlatina, and has been named *scarlatina rheumatica*. Little is known of the pathological anatomy of the affection, since it is seldom fatal.

Symptoms.—These set in suddenly with intense headache and backache and excruciating pains of a rheumatic character, involving many joints and muscles. The temperature rises rapidly, and may reach 106°. Pain at the back of the eyeballs and suffusion of the eyes and face are marked; the mucous membrane of the mouth and throat is congested and swollen, and there is an erythematous flushing of the skin, which produces the short-lived initial characteristic rash.

In a few hours the patient may be unable to stand, or even to lie in bed without tossing about, which greatly intensifies his joint and muscular agonies; hence the popular name of 'breakbone fever.' This stage of acute pain, great prostration, and fever generally terminates about the third morning by a rapid crisis, which produces sweating, diuresis, and sometimes diarrhœa or epistaxis; with the crisis the rash disappears. Occasionally the crisis is gradual; but in either case, during the apyrexial state there is great and marked relief of all the joint and muscle pains—so much so that the patient is generally able to move about without any inconvenience.

In two, three, or four days again he is seized with another short, sharp fever, and return of his joint and muscle pains,

often of more intense degree, and the body is covered with an eruption. This terminal rash remains after the disappearance of the fever, which is generally an affair of hours, terminating in a crisis. The rash is very variable, but characteristic in its polymorphous nature. It begins with itching in the palms or dorsum of the hands and spreads over the body, being sometimes of a roseolar type, at others distinctly rubeolar, erythematous, or even urticarial. It is, however, followed for a few weeks by fine branny desquamation.

As a rule, convalescence sets in after the eighth or ninth day from the initial seizure, but tenderness and joint-pains may remain for some weeks, and even permanent stiffness of the finger-joints may result ; orchitis and enlargement of the cervical glands are common. In some epidemics relapses are not rare. A slight degree of albuminuria is almost constantly present.

Diagnosis.—This is never difficult, owing to the sudden fever, the presence of both an initial and terminal eruption, and the widespread nature of the epidemic. *Acute rheumatism* or *scarlatina* with rheumatic complications may be confounded with it, but not when the remarkably constant presence of the initial and terminal attacks, separated by an apyrexial period, are considered.

Yellow fever has been confounded with dengue, but the almost constant presence of jaundice, severe albuminuria, black vomit, and the slowing of the pulse when the fever is still high, are, when taken together, so characteristic of yellow fever that mistakes can seldom occur, except when both diseases occur in epidemic form in the same place at the same time.

Prognosis.—This is always good, a fatal issue being very rare, save in the case of infants and aged feeble subjects.

Treatment.—Nothing will shorten the attack, and even quinine fails appreciably to influence the temperature. Antipyrin gives some relief, and salicylates may be tried. Belladonna, combined with morphia, hypodermically, always relieves the intense joint-suffering. The patient should be confined to bed from the first onset, and hyperpyrexia treated by cold sponging or the wet pack ; symptoms, such as vomiting, constipation, diarrhœa, &c., the joint-stiffness, and pains during convalescence, are to be treated upon general principles.

DERMATITIS, EXFOLIATIVE

Under this name several distinct skin-inflammations are included, but the title should be reserved for the type of disease described by Hebra and Devergie under the name of Pityriasis Rubra, and later by Erasmus Wilson, who gave

the affection the name of *Dermatitis exfoliativa*, but with the reservation that, though the disease is always a serious one, it is not so generally fatal as these earlier writers found it to be.

Pathology and Etiology.—About its pathology much has yet to be discovered. It begins as a superficial general dermatitis, which produces an abnormal degree of cornification in the epidermic cells, the inflammatory process gradually involving all the tissues of the skin. The infiltration of the new cells and connective tissue ultimately ends in cicatrisation, and leads to atrophy of the papillæ, hair-follicles, sweat and sebaceous glands, and pigmentation when the affection is of long standing. Whether the disease is produced by a microbe in the affected tissue, or by a poison generated within the body, or introduced from without, remains to be proven. The disease is most frequent in males (the four cases seen by the writer were all females), and it generally occurs about middle life.

Symptoms.—Two well-defined forms of general exfoliative dermatitis are met with, usually easily differentiated by the history of their onset. In the so-called *primary* form, the disease attacks a patient who is apparently in good health and free from any cutaneous affection ; in the *secondary* form the general dermatitis arises in the neighbourhood of, or on the site of, an area of eczema, psoriasis, lichen, erythema multiforme, or erysipelas. In either case the affection tends to become general or universal in its distribution, and soon the distinctive characteristics of the original skin-affection are lost or merged in the established general dermatitis.

The disease in the primary form begins as a bright patch of erythematous redness in one or several places at about the same time, which coalesce, and in a few days or weeks the entire surface of the body becomes dry, red, and covered over with large scales resembling flakes or frills of tissue-paper. Upon the face the scales are more or less furfuraceous, whilst upon the palms and soles the epithelium may desquamate in large plaques, as in scarlatina, and even the nails may be shed. The large, dry, papery flakes or scales are produced in abundance and thrown off or detached continually, so that the patient's bed becomes filled with them, handfuls being shed every twenty-four hours. There is sometimes in the acute cases a certain amount of fever and malaise, but many patients exhibit no pyrexia all through their progress. In very chronic cases emaciation, muscular and nervous prostration, and insomnia supervene. It is generally surprising how little itching or evidence of irritation may be present, though in neurotic patients occasionally severe pruritus and tingling may be prominent throughout. There is no secretion, and the bed-linen is not soiled or stiffened, as in eczema ; there

is little thickening or infiltration of the skin, and the redness disappears on pressure.

The condition may last for many months or years, the great majority of cases being slow and chronic in their course. Under treatment the dryness and redness of the skin gradually subside all over the body, and a diminution in the amount of desquamation slowly becomes apparent. Relapses are very liable to take place spontaneously or on exposure to trivial irritation, and after recovery the skin may long remain pigmented and dry.

In cases which do not yield to treatment the emaciation reaches a marked degree, and bronchial or pneumonic troubles, or nephritis, are generally the immediate cause of death.

Diagnosis.—The established disease, whether it has been secondary or primary, exhibits a picture which can hardly be mistaken. The universal or general distribution of the redness and dryness, and the abundant papery desquamation, are quite characteristic.

Eczema, even when extensive, is seldom, if ever, quite universal; and there is always the serous discharge present which leaves its mark on the patient's linen. This may be absent in the scaly or dry stage of eczema, but the history of its presence at an earlier stage is obtainable, and there is marked infiltration of the skin.

Pemphigus foliaceus may be as generally distributed as the affection under consideration, but careful inspection or cross-examination should reveal the presence of bullæ before the stage of desquamation, or the distinct history of them at an earlier period.

Psoriasis is never universal in its distribution in the same sense as desquamative dermatitis is, since there are always portions of healthy skin between the affected areæ, and the silvery plaques of psoriasis consist of a thick layer of superimposed scales. Where psoriasis has been the starting-point of the disease, generally a definite history of the site of a long standing silvery eruption on the knees, elbows, and scalp can be obtained.

There is an *epidemic* form of dermatitis described as occurring in workhouses and infirmaries; it is supposed to be caused by a specific diplococcus, and it appears to possess most of the characters of an eczema, only it is contagious, and it runs a more rapid course than the typical exfoliative dermatitis and, like this latter, is often fatal.

Prognosis.—This is always serious, and in broken-down constitutions generally fatal, especially so in subjects of renal disease and chronic alcoholism. The prognosis is better in young subjects when it runs an acute course.

Treatment.—Internal medication is of little value. The only hope of influencing the disease is by the most patient

and persevering application of a bland unirritating oil or animal fat, which is to be applied several times a day, after warm baths. The different methods of carrying out this treatment are detailed in the author's 'Dictionary of Treatment,' pp. 744, 745, where also Mackenzie's plan of treatment by watery lead solutions is described.

DERMATITIS HERPETIFORMIS, or HYDROA

These names are given to a number of skin-inflammations, formerly described under the names of hydroa gestationis, or pruriginous pemphigus, or pompholyx, dermatitis multiforme, erythema bullosum, &c., and grouped by Duhring under the single title of D. herpetiformis.

The chief feature of the affection is its characteristic polymorphous nature, and Duhring described the different forms of it as erythematous or urticarial, vesicular, bullous, papular and pustular, according to the predominating type of the eruption; but the mixed or multiple form is the most common, in which all or most of the above types exist together.

Etiology and Pathology.—Its causation is obscure, but the affection has been generally regarded as a neurosis, being often found associated with nervous prostration and mental exhaustion. The form occurring in pregnancy is probably the result of some centric nerve-condition started by uterine irritation.

J. C. Johnston, reasoning from the similarity of the eruptions which sometimes follow the administration of quinine, iodides, and bromides, concludes that the etiology of the cutaneous condition under consideration must be accepted as a toxæmia, the toxin being produced within the patient's body. He points out that indican is very frequently found in the urine, which also strongly supports the toxæmic theory, and is further strengthened by the successful results of treatment based exclusively upon eliminatory principles.

As in the case of pemphigus, there is always found a great increase in the eosinophile-cells in the blood, and an abundance of them in the fluid contained in the bullæ. Whitfield found that the eosinophiles are increased from the normal 1·5 per cent. of total leucocytes in the blood to 15 per cent., and to nearly double this percentage in a case of the uterine variety; and Bushnell has proved that the phagocytic power of these cells is diminished. The anatomical changes correspond to the clinical manifestations, being chiefly if not entirely confined to the superficial layer of the skin.

Symptoms.—The symptoms commence in patients of any age, generally with itching, and sometimes there is slight

febrile disturbance, but the general health is usually little affected throughout. The rash first affects the arms, thighs, and neck ; it is bilateral and more or less symmetrical, ultimately involving large tracts of the skin all over the body. Its first appearance consists of raised erythematous patches like erythema multiforme, showing papules which rapidly change into vesicles grouped in small clusters on the margins of the red raised plaques, and at this stage not unlike ordinary herpes. Bullæ form from the vesicles or appear alongside them on the margins of the erythema, or they may arise independently of any erythema. They vary from the size of the vesicles seen in ordinary herpes to blebs of the magnitude of a small plum, and they are usually oval or circinate, and tense.

Wheals like those seen in urticaria are sometimes present. Pustules are often observed forming from the vesicles or bullæ, or containing pus on their first appearance, and surrounded by an area of inflammatory redness ; scabs and crusts form as the vesicles and bullæ dry up. Itching and smarting become severe, and the pustular form may induce severe constitutional disturbance.

Rarely are the mucous membranes involved. The attack may last for several weeks, the eruption extending over the body in successive crops. As the vesicles and bullæ wither, the erythematous base loses its redness, and temporary recovery follows. The disease is, however, essentially chronic ; and after an interval of weeks or months the eruption appears again, to go through the same stages as before. As the morbid condition which causes the affection becomes established, the attacks return at shorter intervals and last longer, till the skin is never free from some manifestation of the polymorphous eruption, which in severe cases may cover almost the entire surface of the body.

The form of dermatitis herpetiformis which occurs in association with pregnancy and childbirth, and which is known as *hydroa gestationis*, and *herpes*, or *erythema*, or *pemphigus gestationis*, occurs after the fourth month, most usually in the later months of gestation.

It does not markedly differ from the multiple or mixed type of herpetiform dermatitis in its characters, but it is much milder, and pustules do not form. The most common type of the polymorphous eruption is the erythematous, associated with bullæ, though the vesicular form occurs sufficiently often to give the name to the disease. Relapses occur, and the disease is very liable to recur with each succeeding pregnancy. The multiform eruption may continue to come out after delivery, and in rare instances it may become established, passing into the ordinary non-puerperal form.

Diagnosis.—Even in a first attack this is not difficult after watching the case for some days. A careful examination

of the eruption on different parts of the body will reveal its polymorphous characters, whilst each successive relapse renders the diagnosis more certain.

Pemphigus is a purely bullous eruption; the blebs do not show an erythematous base, and there are no vesicles, pustules, or wheals present. As a rule, itching is seldom a marked feature, and the disease yields to arsenic. In the acute variety there is considerable fever, and in the chronic severe exhaustion. In *foliaceous* pemphigus the blebs are never tense; the contents are always turbid or purulent, and the progress is towards a fatal issue.

Erythema multiforme, when recurrent, often presents polymorphous characters, but bullæ are rare, and pustules seldom if ever seen; itching is generally absent or trivial. The predominating lesion is always erythematous, and the individual attacks or relapses run a much shorter course.

Eczema rarely approaches in appearance the disease under consideration, having its vesicles always smaller, and never clustered or grouped, and the red weeping surface is characteristic.

Herpetiform impetigo is a rapidly fatal and rare disease, occurring in pregnant women. The pustules are very small, situated upon an inflamed base and spread peripherally, soon covering the entire body with crusts, and often involving the mucous surfaces, though there is but little itching.

Urticaria, when it appears in its rare *vesicating* form, is generally met with in children. The rapidity of its course, its short duration, and the absence of marked polymorphous characters, as a rule easily distinguish it. It must be remembered, however, that D. herpetiformis sometimes occurs in children.

Prognosis.—As a rule the disease is seldom fatal, save in those cases where pustules are numerous; but the prognosis is always serious as regards the probability of relapses. The tendency of the disease is to return and ultimately to become chronic or permanent, though it may interfere little with the general health. Occasionally it disappears spontaneously, and this is most likely to occur in young subjects and in acute cases. The ultimate prognosis in pregnancy cases is good.

Treatment.—This is unsatisfactory, and little can be done save in the direction of relieving the itching and controlling the severity of the superficial inflammatory symptoms. All errors in feeding and everything wrong in the environment and habits of the patient should be corrected; a milk-diet, rest in bed, and saline purgatives may be prescribed during the acute stages of the attacks. Johnston, as already stated, maintains that the chief indication for treatment lies in the elimination of the toxic agent, which he effects by saline diuretics, copious and continuous draughts of water, and

lavage of the colon. Internally, arsenic has some efficacy, especially in the chronic stages; and Crocker believes belladonna in full doses to be useful. Ichthyol in large doses is recommended by Pringle. Itching and insomnia may be relieved by antipyrin and chloral; morphia often aggravates the former symptom. Engman, as quoted by Walker and Gardiner, has reported a case in which iodide of potassium always caused the eruption to reappear. Locally the treatment is that suitable for acute eczema—viz. weak lead and tar lotions or ointments. The constant use of warm baths has its advocates, but Duhring recommends a strong sulphur ointment (2 drs. to 1 oz.) to be rubbed in so as to break up the vesicles, bullæ, and pustules.

DIABETES INSIPIDUS

This, known also as Polydipsia, is a disease characterised by the persistent secretion of a large amount of urine of low s.g. free from albumin and sugar.

Etiology and Pathology.—It is usually met with in males under ten years of age, and between puberty and middle life. Often there is a neurotic history, and heredity plays an important part in some cases, the disease passing through several generations. Diabetes, albuminuria, early syphilis, tuberculosis, and gout are often present in the parents of young subjects suffering from the affection. In a considerable proportion of the cases there is a history of a previous concussion or head-injury. In others there is a history of malnutrition, mental worry, influenza, or the presence of a cerebral growth may be elicited; sometimes the disease has been found to follow injury to the abdomen which involved the solar plexus.

Its pathology is probably a neurosis producing a failure in the inhibitory control of the vaso-motor nerves of the kidney. Peripheral irritation of the abdominal plexus or centric stimulation of the floor of the fourth ventricle has produced the condition in animals, and section of the great splanchnic is followed by an excessive flow of weak urine. The enlargement of the kidney, and dilatation of its pelvis, found after death are obvious results of the hyper-secretion of urine.

Symptoms.—The two main characteristic symptoms are the increased flow of urine and the excessive thirst. The total amount of urine in the twenty-four hours may reach three or four gallons; in a case recorded by Trousseau, the amount reached nine and a-half gallons. Twelve to sixteen pints are often voided in marked cases. The s.g. is always low, and the writer found in one case that it frequently

registered 2° below that of distilled water when taken warm from the bladder, rising to 1002 on cooling to the ordinary temperature of the ward ; the total amount was twenty-four pints daily.

The total amount of solids is often slightly increased above the normal average. Albumin in small amount is sometimes present towards the end in grave cases ; as a rule, it is absent. Traces of sugar and inosite are occasionally noticed. The colour of the urine is pale with a faint yellow or green tinge, not unlike what might be produced by adding a little ascitic fluid to a large volume of water. It generally exceeds the total amount of the liquids consumed, but in the later stage falls short of this. Thirst is excessive : often this symptom is more distressing than the thirst in saccharine diabetes ; and in the earlier stages sometimes the appetite may be voracious. The mouth and tongue are continually parched, the skin is dry, the bowels constipated, and the temperature is constantly subnormal. Pains in the back and loins are common, headache is not infrequent ; loss of the tendon-reflexes, insomnia, impotence, and dimness of vision are generally noticed. The patient becomes restless and irritable, and the mental processes sluggish or weakened. In grave cases marked emaciation, with great muscular weakness, compels the patient to take to his bed ; his appetite fails, diarrhœa sets in, and he gradually passes into coma, or succumbs to a low form of pneumonia, or dies from interference with the vital functions caused by the presence of cerebral growths in those cases where the disease is of centric origin. In mild cases there may be little interference with health or comfort, save in the worry of the frequent micturition. The disease may last for a great number of years, and in a considerable number of cases it disappears spontaneously.

Diagnosis.—*Diabetes mellitus* is easily excluded by the low s.g. and the absence of sugar, or by the mere occasional presence of a trace of sugar. *Hysterical hydruria* is recognised by its marked intermittent or transitory character, and by the association of hysterical symptoms. *Hydronephrosis* and *cystic disease* of the kidney lead to the passing of large amounts of pale urine, but the intermittent nature of the polyuria is always marked, a tumour can be detected, and the examination of the urine shows diminution of total solids excreted. *Chronic Bright's disease*, with contracted kidneys, may closely simulate D. insipidus. The distinguishing points are : (1) there is always some albumin present in every case, though it may occasionally disappear for a day or two ; (2) the total excretion of solids is diminished ; (3) hyaline casts are usually present ; (4) there are evidence of arterio-sclerosis, hypertrophy of the heart, and increased arterial tension, and sometimes retinal hæmorrhages and slight œdema. *Lardaceous kidney* is

recognised by the history of suppuration or of syphilis, the presence of albumin and casts, and the evidences of the amyloid degeneration in the liver and spleen.

Prognosis.—This is difficult, and, as a rule, the case must be under observation for a considerable period before a forecast can be made. Where an obvious cerebral lesion is the cause of the polyuria, the prognosis is always grave. Where the disease follows cranial injuries, recovery generally supervenes. Acute cases associated with early deterioration of nutrition and innervation often end fatally, whilst those more chronic cases beginning without any obvious cause (the so-called idiopathic cases) often persist without any injury to health for many years, and may ultimately recover completely. In a small percentage of cases the disease becomes identical with renal inadequacy, and the patient dies from uræmia.

Treatment.—The list of drugs credited with a curative action in this disease is a long one, but there is no evidence that the pathological factor can be modified by any of them. The treatment, therefore, resolves itself into the relief of symptoms, and often must be pursued upon purely empiric lines. Trousseau recommended valerian in heroic doses, but the general experience is that in amounts capable of diminishing the polyuria it always upsets digestion and appetite. Perhaps the best results have been obtained by full doses of antipyrin alternating with short courses of nitroglycerin. Bromides, phosphorus, morphine, belladonna, pilocarpine, ergot, salicylates, strychnine, and salts of gold, silver, and arsenic, have been tried with varying success. Electricity should be always tried, notwithstanding that it generally fails, because several cases seem to have yielded to it. The constant current may be used with one pole on the occiput and the other over the stomach and loins, or the current may be passed through the bulb or base of the brain by placing the positive pole on the occiput and the negative on the front of the spine in the region of the naso-pharynx, as recommended by Robertson.

Reduction in the amount of liquids consumed can only be safely carried out to a small extent, and the diet should be generous and varied, with a large proportion of carbohydrates. Change of air and scene, with absolute avoidance of severe mental work, often effects more than drugs. Alcohol in all forms is generally injurious. Insomnia, constipation, and indigestion call for the recognised palliatives.

DIABETES MELLITUS

This is a disease in which sugar in the form of grape-sugar or glucose persistently appears in considerable quantity in the urine, which is nearly always markedly increased in amount. The term *glycosuria* is usually reserved for those cases where sugar is present in small quantity, for the most part in an intermittent way, and where the amount of urine is not increased. There is, however, no hard-and-fast line between the two conditions, though generally they are so easily distinguished clinically, because in a small percentage of the so-called cases of intermittent glycosuria the presence of sugar becomes persistent and the condition passes finally into diabetes.

Etiology.—The disease appears most frequently in males; and though no age is exempt, it occurs oftenest about the ages of forty or fifty years. It is much more common in cities than in rural districts, being notably frequent in Jews and almost unknown in negroes; it is prevalent in India amongst the Hindus, and rare in China and Japan.

The cause of the disease is unknown, though amongst the etiological factors the following have been considered operative—viz. heredity, obesity, excessive use of sugar and alcohol, prolonged mental anxiety, brain over-work, injuries (especially head-injuries) and intracranial growths, Graves' disease, influenza, and the exanthemata. A considerable percentage of cases are associated with pregnancy, while gout certainly plays an important part in the milder forms of glycosuria. A microbic origin has been surmised in those cases where a husband and wife have been found suffering from the disease.

Morbid Anatomy.—The most obvious changes found in the bodies of diabetics are due to the extreme emaciation present; in the great majority of cases any changes in the liver and kidney are those of an enlargement or fatty degeneration, which is a secondary and not a primary lesion. Various forms of nephritis have been described, but these are not constant; and the same must be said of the degenerations and changes in the renal epithelium in the tubes and loops. In an inconsiderable number of cases changes in the medulla and growths in its neighbourhood are noticeable, but no constant nerve-lesion is found. Tubercular or broncho-pneumonic lung-changes are common. The blood may contain several times the normal amount of sugar, fat is often found in abundance in the plasma, and fat-embolism of the capillaries of the lung is not uncommon. Glycogen has been demonstrated in the polynuclear leucocytes.

Gross changes in the pancreas are found in more than half of the subjects dying from diabetes, and these lesions justify

the conclusion that the pancreatic change is primary and is the cause of the disease in these cases, and not merely a secondary phenomenon, as is the likely explanation of the secondary changes found in the other viscera, in the blood, and in the spinal and sympathetic systems of nerves. The diseased condition of the pancreas is evidenced by atrophy, cirrhosis, fatty enlargement, cancer, calculi, &c., the most important change found being the interacinar type of chronic pancreatitis in which the islands of Langerhans are involved.

Pathology.—The pathogeny of diabetes still remains a vexed question and a fruitful source of conflicting theories, none of which can yet be said to meet all the requirements of a good working hypothesis, and it is probable that there are different varieties of diabetes, each with its own special pathology.

In health, Bernard and his followers maintain that the saccharine and carbo-hydrate elements of the food after digestion are stored in the liver in the form of glycogen, which is again leisurely converted into grape-sugar or glucose, and is gradually let into the blood-stream, where it is steadily destroyed to meet the requirements of the system. (The muscles have the same power of storing glycogen.) To explain the phenomenon of diabetes, three main theories have been advocated:

1. That the liver fails to stop the altered carbo-hydrates and sugar from obtaining an immediate entrance into the general circulation; the result is that a large amount of glucose appears in the blood and is excreted in the urine, the primary error being a breakdown in the normal *sugar-assimilating function*. This theory is still held by Pavy. He, with certain modifications, maintains that in health the intestinal villi also stop the carbo-hydrates and convert them into fat; and consequently, when the liver and villi fail in their sugar-assimilative function, sugar accumulates in the blood, and diabetes results. This hypothesis affords an explanation of the milder or so-called 'hepatogenetic,' 'lipogenic,' 'dietetic,' or 'alimentary' form of diabetes, in which the sugar disappears from the urine upon restricting or stopping carbo-hydrates in the food. It does not, however, explain the severe forms of diabetes in which sugar continues to be present in the urine when all carbo-hydrate food is excluded from the patient's diet. In these cases sugar is manufactured from the proteids of the food and from the patient's tissues; and to afford an explanation of this phenomenon, Pavy is driven to the conclusion that the sugar is manufactured from the food and tissue proteids by the aid of a ferment which, he theorises, exists somewhere in the system. In these cases of so-called 'composite' diabetes, acetone, and other products, the result of the proteid disintegration of the food or tissues, appear

along with the sugar in the urine and explain the tendency to diabetic coma.

2. Many authorities maintain that the explanation of diabetes is to be found in *an increased formation of sugar* in the system, probably through excessive action of the glycogenic function of the liver. The excess of sugar is then poured into the circulation more quickly than it can be burned up or destroyed by the tissues (especially by the muscles) in order to maintain the body-temperature. It is here that the different nervous theories of the origin of diabetes come in—viz. vasomotor dilatation of the hepatic capillaries, puncture of the floor of the fourth ventricle and medulla, &c., and cerebral growths, the central nervous irritation leading to a greatly increased flow of blood through the liver.

3. The view is gaining ground that diabetes is the result of *diminished sugar-destruction.* By what means the glucose poured into the blood-stream from the hepatic vein is broken up, we can only conjecture. Theoretically, a failure or diminished activity of the combustion-process in the lungs or tissues would meet the difficulty. Hence the Bunge theory shifts the site of the mischief to the muscles. It is, however, at this point in our difficulty that the pancreatic theory comes in. Lépine and others believe that the pancreas furnishes an internal secretion, and Schäfer believes that the parts of the gland which manufacture this secretion are the columns of polygonal cells of the islands of Langerhans embedded in its tissue. If the theory be accepted that this internal secretion, when it enters the blood, possesses the power of breaking up the sugar which it finds there, the best working hypothesis of the pathology of diabetes is afforded. Whether the agent which accomplishes this glycolytic change is of the nature of a ferment as maintained by Lépine, or of the nature of a simple chemical product somewhat analogous to the active principle of the internal secretion of the thyroid gland, is not important. Neither is it of primary importance whether or not this substance, as believed by Kaufmann, acts by influencing the sugar-forming function of the liver—a theory which shifts the influence of the pancreas from being a factor in the diminished destruction to that of a cause of increased production of sugar.

The pancreatic theory is the only one which harmonises with the morbid anatomy and with recent experimental results.

When the pancreas is *entirely* removed in animals diabetes results; and that this is not caused by injury to the abdominal nerves during the operation of removal is demonstrated by the fact that if a portion of the excised gland be grafted amongst the abdominal muscles outside the peritoneal cavity, no glycosuria follows unless the graft necroses, as shown by Minkowski.

Considerable interest has been aroused by the discovery that the mucous membrane of the duodenum secretes a substance to which the name of 'prosecretin' is given; this in its turn is changed into secretin under the influence of the free HCl from the stomach; and it is held that the secretin acts as a chemical excitant or stimulant to the pancreas, and causes the organ to manufacture both its external and internal secretion. The pathology of the so-called phloridzin diabetes is explained by the action of this glucoside in so altering the renal epithelium as to permit of the passage outwards of the sugar in the renal vessels.

Symptoms.—These vary in the acute and chronic forms of the disease, but they are essentially the same, differing only in degree. The acute form of the disease generally is seen in young subjects, and the chronic in obese subjects about middle life.

The first symptom often is excessive thirst, followed by polyuria, muscular weakness, increase of appetite, and emaciation. The onset may be sudden or insidious, and in the severe cases emaciation and weakness appear early, whilst in the chronic form these are not evident for a considerable time. In many instances advice is first sought for some of the complications of the disease, as eczema, carbuncle, gangrene, impotence, or cataract. The patient often complains of a sweet taste in his parched mouth, and commonly the breath has a characteristic sweet odour; the tongue is dry, red, and abnormally clean; the cheeks are often flushed, though the temperature is below the normal; the skin is dry, and the thin hair feels like dry flax; as a rule, the bowels are constipated. In acute untreated cases the urine may measure up to sixteen pints in the twenty-four hours; in the more chronic forms it may not exceed five pints; in both cases micturition is frequent, and sleep considerably disturbed by the repeated emptying of the bladder.

The persistent presence of sugar and the augmentation in the bulk of the urine are the primary symptoms of diabetes; though sometimes the volume of the secretion in otherwise well-marked cases may never exceed sixty ounces. The fluid is usually pale amber: sometimes a greenish tint is present; its s.g. varies from about 1025 to 1045; it is usually clear, and often a sweet apple- or hay-like odour is obvious. It is nearly always acid in reaction. When the secretion is very abundant the s.g. may never exceed 1015 or 1020.

The amount of sugar, which is always glucose (grape-sugar), ranges within wide limits from ·5 to 12 per cent., and the daily quantity may reach as much as 7,000 grains or one pound; in rare cases, twice this amount has been recorded; eight ounces are very often excreted. The quantity is always markedly increased after the ingestion of sugars and starches, whilst the

total urea falls proportionately. Less is excreted during the night, and the morning urine may only show a small percentage.

Tests.—Fehling's solution is the most frequently employed test; it depends upon the reduction of cupric sulphate in hot alkaline solution by glucose to the condition of the yellowish or red cuprous oxide; but *it fails in ammoniacal urine*: (1) 34·64 grms. of pure sulphate of copper are dissolved in 500 c.c. of distilled water; (2) 60 grms. of solid caustic soda and 173 grms. of Rochelle salt are dissolved in 500 c.c. of distilled water. The solutions should be kept separate till about to be used, as they decompose when they are kept mixed for any length of time.

About half a drachm of each solution should be mixed in a test-tube and heated to nearly boiling-point and a few drops of the urine added, when a yellow precipitate, quickly changing to brick-red, will appear at the surface. If less than $\frac{1}{2}$ per cent. of glucose be present the precipitate does not appear till cooling occurs. Boiling is unnecessary and undesirable, as then the presence of glycuronic, uric, and other acids may give the reaction.

Pavy's test-solution is a modification of the above, but the writer always employs it in the more convenient form of dry pellets. Trommer's test is applied by adding a few drops of a copper-sulphate solution to a drachm of urine in a test-tube, and then adding half as much liquor potassæ, when the sulphate is reduced on boiling to the cuprous oxide.

Moore's test of boiling the urine with an equal bulk of liquor potassæ is unreliable. The bismuth, methylene blue, and picric-acid tests possess no special advantages.

Krismer's safranine test is relied upon by Bedford as the simplest and surest of all tests. It is a brownish-red coal-tar dye, and it is used in the following manner: equal parts of a 1 in 1000 watery solution, liquor potassæ and urine are mixed in a test-tube and boiled; the glucose changes the opaque red colour into yellow, if even $\frac{1}{10}$ per cent. only be present. The presence of chloroform and chloral in the urine will reduce the copper salt in Fehling's solution, but not safranine.

Phenyl-hydrazine is the most delicate of all tests for glucose; it is too delicate for ordinary use, since it may give its reaction with the traces of glucose always present in normal urine. About 10 grs. phenyl-hydrazine hydrochloride are placed in a test-tube with twice as much powdered acetate of sodium, and the tube half-filled with urine and boiled for several minutes; the yellow precipitate, on cooling, shows under the microscope the needle-shaped crystals of glucosazone arranged in fans or stars.

The fermentation-test, except for its slowness, is the most valuable of all tests, and it affords the best ready method of

roughly determining the quantity of sugar present. With the following precautions it is infallible: the dry yeast should be tested by the addition of a few drops of tincture of iodine to prove its freedom from starch; in case of doubt its activity should be demonstrated by a control experiment with a solution of glucose; and the diabetic urine should be acidified, if necessary, by a little acetic acid.

The s.g. of the urine is taken, and a piece of yeast the size of a large pea is added to a large test-tube completely filled with the urine; the tube is then inverted over a small dish containing a little of the urine, and the whole left in a warm place (90° F. is the best temperature). In twelve to eighteen hours the glucose present is all converted into carbonic acid, alcohol, &c., the gas accumulating at the top of the tube. If the s.g. be taken after cooling, every degree of s.g. lost during the fermentation will indicate 1 gr. of glucose per ounce; and by multiplying this by the total number of ounces passed in the twenty-four hours, the gross output of sugar is roughly and readily determined. Thus, suppose 200 oz. of urine be passed of a density of 1040, and a sample of the day's collection after fermentation and cooling shows the s.g. of 1020, the amount of sugar may be taken as 4,000 grs., or 9 oz. approximately. The tests of Fehling and Ültzmann's polarising saccharimeter are much more accurate and reliable for quantitative analysis.

Acetone in diabetic urine is indicative of serious danger, though it cannot be considered to be the cause of the coma so commonly occurring. It is detected by Legal's test. A little nitro-prusside of sodium solution is added to a small quantity of urine rendered alkaline by liquor potassæ. A cherry-red colour appears, which soon vanishes; the addition of a few drops of acetic acid produces a deep violet colour.

Aceto-acetic, or *diacetic acid* appears only along with acetone, and its presence is of more importance than the mere presence of acetone. It is recognised in *fresh* urine by the addition of a few drops of ferric chloride solution which precipitate the phosphate of iron; after filtering the urine, a few more drops of acetic acid strike a deep Burgundy-red colour. Salicylic and carbolic acids and antipyrin give the same reaction; but if the urine be boiled before adding the ferric chloride, the colour is not produced by diacetic acid, as this substance is driven off by the heat.

Beta or *hydroxybutyric acid* is of more serious importance, since it is held to be the cause of diabetic coma by its power of lessening the alkalinity of the blood; unfortunately no clinical methods are available for its detection save such as necessitate distillation or the use of the polarimeter. If diacetic acid is not present, the absence of this acid may be inferred, as they always occur together.

Albumin appears in the urine in a considerable number of cases, and when abundant indicates secondary renal trouble. It is commonly present in coma, and casts also are then found. Pus and mucus are generally in evidence in the urine of diabetic women from irritation of the vagina and vulva. Urea is generally increased, and so are the other constituents, notably uric acid, lime-salts, and ammonia.

The blood in diabetes always contains a large increase (0·25 to 0·55) in its normal (0·1) percentage of sugar ; fat is often present in abundance, and the alkalinity is considerably reduced in all severe cases. Nervous symptoms are present : neuralgias, cramps, and pains in various regions, the result of multiple neuritis, sometimes ending in paralysis and wasting of muscles ; the knee-jerks are often absent (the obturator, crural, and peroneal nerves are most frequently affected in diabetic neuritis), and ataxic symptoms may develop ; the mental process is frequently sluggish or perverted, the temper irritable, and the special senses dulled ; loss of virility is common, and the menses are usually suppressed.

Amongst the numerous *complications*, the most serious is coma, which will be described later on. Various skin-affections, as erythema, pigmentation, eczema (especially of the genitals in women, which causes distressing pruritus), balanitis, carbuncles, and boils, or cellulitis are not infrequent. Gangrene is common in aged diabetics, especially affecting the feet and legs ; it is usually of the dry variety, and perforating ulcer of the foot sometimes occurs. Pulmonary complications account for a considerable percentage of deaths, as tuberculosis and pneumonia are rapidly fatal in diabetes, and often insidious in their onset ; pulmonary gangrene may also occur. Eye-complications are common, cataract being the most noticeable ; loss of accommodation, retinitis of a characteristic type producing the condition known as diabetic amblyopia, and optic atrophy may occur. Ear-inflammations are serious, and rapidly end in bone-mischief. The heart in the later stages shows weakness, causing œdema of the legs and breathlessness ; and a small percentage of cases develop renal disease.

Digestive troubles are rare till the later stages of the disease, but caries of the teeth and ulceration of the gums are common ; dyspepsia, the result of the restricted diet, is troublesome in a small percentage of cases. It is a fact to be remembered that often the sugar disappears from the urine during some acute febrile or other complication.

Coma may occur in any case of diabetes, but it is much more common in the severe form which occurs in young subjects, and in these it is the usual mode of termination. In children it may appear within a week or two of the first symptom of the disease ; the writer has seen it supervene upon the fourth day in one case. In chronic cases it may not show itself till

years after the onset of the glycosuria. It has been so often noticed to follow the institution of a very rigid diet, or the sudden stoppage of the imbibition of water, that many authorities regard the dietetic change as an important factor in its cause ; but upon the other hand, it has been associated with a free indulgence in carbo-hydrates. Mental strain, powerful emotional excitement, prolonged muscular exertion, and obstinate constipation have been considered as exciting causes.

Several types of diabetic coma have been met with, and Frerichs described three ; but the difference is probably one of degree. The onset in rare cases may be so sudden as to justify the term 'explosive'—great restlessness, rapidity of pulse, and active delirium, with deep, laboured breathing, rapidly passing into profound coma and death. In other cases the attack is less sudden, being preceded by a marked diminution in the quantity of urine and sugar excreted. The general condition becomes one of extreme collapse, with coldness of the extremities and subnormal temperature, the heart-failure obviously playing the chief part, as evidenced by the small weak pulse and the lividity ; drowsiness soon appears, which steadily passes into coma. This type is seen oftenest in obese diabetics past middle life. More usually the premonitory symptoms of gastric disturbance and pain, with rapidity of pulse and dyspnœa, may last for a few days before the advent of marked drowsiness and coma. This latter is often for a day or two incomplete, during which the patient may be roused to answer a simple question or to sit up in bed. The condition of the respiration often corresponds to that described by Kussmaul as air-hunger, in which there is deep and sighing breathing, both inspiration and expiration being laboured, without marked frequency. The quickness of pulse, drowsiness, and dyspnœa should be always regarded as grave symptoms, and life seldoms lasts beyond thirty-six to forty-eight hours after coma has developed. Sometimes before death the subnormal temperature rises 5° or 6° F. above the normal.

The breath usually has a fragrant or chloroform-like odour, and a similar smell is detected in the scanty hyperacid urine, which generally contains albumin, and often casts. The odour depends upon the presence of acetone, which may be present for some days before the attack. Legal's test or Lieben's iodoform-test shows acetone to be present ; and Gerhardt's Burgundy-coloured reaction is obtained by adding ferric chloride, demonstrating the presence of diacetic acid. These two substances are not the cause of the coma, since they can be administered in large doses without inducing stupor ; they are oxidation-products of β-oxybutyric acid, which is believed by many authorities to be the real cause of the coma, and large amounts of this acid are excreted in the urine and

found in the blood and tissues after death. The entire phenomenon of diabetic coma is obviously an auto-intoxication, and probably renal inadequacy plays an important part in it as well as β-oxybutyric acid ; hence it is still supposed by a few to be a uræmic process. Fat-embolism and ptomaine-poisoning have been suggested as probable causes. In some cases cardiac failure is an important agent, and it is stated that in this variety acetone-products may be entirely absent.

From the above description it will be seen that the progress and duration of diabetes are exceedingly variable. In middle-aged obese subjects the disease may last for many years, though death generally occurs inside three or four years. In one well-marked case under the writer's observation, a quarter of a century has elapsed since the disease appeared, the sugar only a few times disappearing under the most rigid dieting, and the patient is still enjoying fair health, though living upon a diet almost devoid of carbo-hydrates. In young subjects and children and in lean patients the course may be extremely rapid, death occurring in a few weeks or months.

Diagnosis.—This rarely gives rise to difficulty. The constant presence of glucose in the urine is distinctive. Mild cases of intermittent glycosuria, especially in gouty subjects, should not be mistaken for diabetes. Williamson's test may be applied to the blood in any doubtful case of diabetes : it depends upon the power of a drop of diabetic blood to discolour a weak alkaline solution of methylene blue when heat is applied. Bremer finds diabetic blood-films do not stain with Congo-red. These tests are never necessary in clinical work, but they may be resorted to in suspected frauds in connection with life assurance. It is necessary to bear in mind that a not inconsiderable number of diabetics seek advice in the early stages of the disease for some of the complications, as boils, pruritus, amblyopia, muscular weakness, &c.

Prognosis.—As already stated, this is always grave in children and young subjects, and in them the disease runs a very rapid course little influenced by treatment. In older and fat subjects the duration is longer, though the disease is seldom, if ever, curable in the strict sense of the term. The prognosis is always bad when the sugar fails to disappear under a strict diet. Acetone-products in the urine deepen the prognosis, and premonitory signs of coma justify a fatal forecast. The point in the prognosis to which the writer attaches the utmost importance in the majority of the cases of diabetes met with in private practice, is a study of the character of the individual as regards his will-power in adhering to a strictly regulated diet. A few interviews will often convince the physician of the absolute hopelessness of treating some patients ; especially is this seen amongst women. Poverty is a most serious element in the prognosis. Pneumonia, tubercle,

gangrene, and carbuncle seriously influence the progress of the disease, tending to cause sudden collapse.

The presence of albumin in any marked amount is serious, and especially so if casts are found.

Treatment.—Before submitting a patient to any treatment the urine should be collected for one or two days, whilst the ordinary diet is being used, so that an estimate may be formed of the amount of sugar voided, and thus of the severity of the case. After this, sugar should be absolutely forbidden in any form, and other carbo-hydrates much restricted, and in a few days entirely stopped. Many mild cases require little further treatment, the sugar entirely disappearing as long as the carbo-hydrates are avoided. The physician, however, after a week or two, should permit a little bread or baked potato, gradually increasing the quantity till the sugar returns, so as to ascertain the patient's power of assimilating or tolerating starchy food, as it is desirable to give him any amount short of that which induces glycosuria. Such cases belong to the mild variety of the disease, and require no further management save attention to the laws of health. If the sugar persists after the entire withdrawal of starchy food, the case is a severe one ; and the prohibition must be maintained unless the urine shows the presence of acetone or diacetic acid, in which case a little bread must be permitted, and this may be necessary even in the absence of these products in the urine if the wasting is marked and continuous. Every case must be considered and treated upon its merits, constant trials of body-weight and examination of the urine affording the basis for the increase or diminution of the sugar-forming elements in the dietary.

The main difficulty experienced is in the prohibition of bread, as the substitutes for this are unpalatable and often indigestible. Gluten and almond bread, and cakes made of bran by Camplin's method, are the most frequently employed. Gluten flour is often used ; but many samples of it and of other diabetic foods are fraudulent, and sometimes contain as high a percentage of starch as ordinary white bread : hence it is very necessary that only those sent out by makers of known repute should be used. Brown bread or white, cut into slices and thoroughly toasted or torrified, may be occasionally permitted when the almond or gluten preparations become distasteful.

(In the author's ' Dictionary of Treatment,' on pages 199-209, will be found details of various dietaries, as those of Pavy, Roberts, Ebstein, and Düring, which need not be here repeated.)

The following articles are *allowable* : Butcher meat (liver excepted) of all kinds, poultry and game, fish of all kinds, tongue, ham and bacon, cheese, cream, cream-cheese, butter,

suet, oils and fat, eggs in any form, nuts as filberts, hazel, and walnuts, Brazil nuts, and almonds; cucumber, mushrooms, all green vegetables as spinach, curleys, lettuce, mustard and cress, watercress, and green cabbage with the white portions rejected, Brussels sprouts and broccoli, lemons, pickled walnuts, onions, and mushrooms; all soups, beef-tea and beef-essences and broths prepared from butcher meat and poultry, if not thickened with farinaceous materials; jellies, custard, and isinglass devoid of sugar. With these the recognised bread-substitutes made from gluten, almond, bran, aleuronat, and cocoa-nut.

The following are *forbidden*: Sugar and all saccharine substances, as honey, &c.; bread and biscuits of all kinds, save those already mentioned; all farinaceous substances as flour, meal, macaroni, vermicelli, arrowroot, rice, sago, tapioca, semolina; all sweet fruits, fresh or dried, or preserved, as jams or fruit-jellies; most vegetables which are white, as celery, turnips, potatoes, vegetable-marrow, Spanish onions, parsnips; also beetroot, carrots, peas, beans, and chestnuts; pastry and puddings, and soups of all kinds made with farinaceous materials; oysters, mussels and cockles, and the soft parts or pudding of lobsters and crabs, though the muscle-meat of these may be eaten; liver of all animals is forbidden.

Of liquids, the following are *allowable*: Water, plain or aërated, tea, cocoa-nibs, coffee, and lemonade made without sugar, acid buttermilk, dry sherry, hock, and all *dry* wines; claret, burgundy, chablis, brandy, and whisky in small quantities.

The following are *forbidden*: Milk, cocoa and chocolate, beer, ale, and porter, all sparkling wines and sweet wines like port, tokay; sweetened gin, rum, liqueurs of all kinds, and aërated sweetened waters; glycerin and saccharin may be used for sweetening purposes.

The drug-treatment of diabetes is most unsatisfactory. Opium, morphine, and codeine certainly exhibit considerable power in diminishing the amount of sugar; they should only be given in the severe cases when the sugar remains after the cessation of carbo-hydrates, and when the sugar finally disappears the dose should be diminished, and finally withheld. Of other drugs an enormous list is published, each member being credited with curative virtues on very slender evidence; next to the opium series, antipyrin comes first, with other members of the same group. Salol, salicylate of soda, and bromides appear to have a beneficial effect. Undoubtedly the alkaline spas, especially Carlsbad, Vichy, and Vals, give excellent results; but the bread-substitutes at Carlsbad are as a rule not very palatable.

Pancreatic extracts have failed to realise the hopes at first entertained of them. Recently attempts have been made

to treat the disease by the administration of an acid extract prepared from the duodenal mucosa.

Of the complications, the treatment is generally that of the glycosuria, but the coma deserves the most energetic drug-administration. As soon as the β-oxybutyric derivatives are found in the urine, alkalies should be freely administered ; and if drowsiness supervene the dose should be increased and pushed, even in the presence of coma, till as much as 4 oz. bicarbonate of soda be administered in twenty-four hours. It may be given by mouth, rectum, or veins, or by deep parenchymatous injection in very dilute solution. Citrate of potassium in large draughts of water has proved useful, and by the energetic administration of these alkalies life has been much prolonged, the attack of coma having been entirely removed in a small number of cases.

DIARRHŒA

This is the term used to indicate the condition in which frequent loose or liquid evacuations are discharged from the bowel. It is not a disease, but a symptom of disease, as jaundice, vomiting, and dropsy are symptoms of various affections ; but in these latter cases the underlying conditions are generally easily made out: not so in diarrhœa, which may be the only obvious symptom ; hence many authorities still think and write of diarrhœa, as if it were a disease *per se*, under the name of 'enteritis.'

Pathology.—The modern tendency to regard diarrhœa as a synonym for enteritis leads also to confusion of thought. As our knowledge of the action of purgatives becomes more complete, the confusion and the difficulty of classifying the different forms of diarrhœa will probably grow less, and we may be enabled to divide them into two great groups—one of which would be diarrhœas which depend upon structural or inflammatory disease of the bowel, and the other would include those examples of the condition which are caused by the presence of purgative substances introduced from without or manufactured within the body.

Purgative substances may act in three ways :

1. By simply increasing peristalsis ; the nervous mechanism acting upon the muscular apparatus so that the contents of the canal are hurried on, preventing absorption of the intestinal secretion. This explanation will meet those cases where diarrhœa is the result of mechanical irritation produced by indigestible foods, precipitated casein, &c., as well as those in which the watery motions are the result of a chemical irritant manufactured in the bowel, especially in

the large intestine ; in the same manner most laxatives and medicines of the aloes and cascara type act.

2. A substance may cause purgation without marked increase in the peristalsis, by simply stimulating the glandular apparatus to pour out increased secretion, as is seen in the action of sulphate of magnesium. The chemical products of many fermentations, and the toxins manufactured by microbes, mixed accidentally with the food before ingestion, or developing afterwards in the intestinal tube, act certainly in this way, and cause severe diarrhœa. Such examples of a purely purgative action cover a large percentage of the cases of diarrhœa met with in practice. The over-stimulation of the glandular apparatus leads to the inflammatory changes met with on *post-mortem* examination, which are often erroneously catalogued as the cause of the diarrhœa, whilst they are but the result of the irritation produced by the specific irritant.

3. Purgation or diarrhœa may be caused by the action of a drug, or of a chemical product manufactured in the bowel, which would simply prevent the absorption of the normal quantity of intestinal secretion, always poured out in health, into the canal, peristalsis and secretion not being interfered with or augmented. About this last action less is known than of the other two, but it is highly probable that it is operating in every case of severe diarrhœa caused by the action of irritating products, which lessen the absorbing power of the mucous membrane through the congestion and irritation which they produce. It is probably the explanation of the diarrhœa so common in hepatic and valvular lesions.

From the above point of view, the reading of the great majority of cases of diarrhœa is simplified. Thus the so-called '*nervous*' and 'morning' diarrhœas are evidently caused by an exalted peristalsis of the entire tube from mental or psychical causes. The 'microbic' variety probably corresponds to the vast bulk of all cases of diarrhœa, if we include in it those examples, the result of ferments, organised or unorganised, which pervert the normal process of digestion and lead to the formation of irritating products which cause purgation. To this class belong the summer diarrhœa and the fluxes so common in hot climates. In some cases the micro-organisms, or ferments, have been already acting in the food before it has been swallowed ; in others they only become active in the small intestine, where the resulting toxins and ptomaines, in addition to their severe purgative or irritating qualities, may be absorbed and produce fatal constitutional intoxication. It is also probable that the irritants may interfere with the action of the billions of microbes which in the normal condition are present in the alimentary canal.

The so-called 'lienteric' diarrhœa, in which portions of undigested food are visible in the motions, is probably microbic, but it is conceivable that it may be simply the result of a dyspepsia which permits the food to leave the stomach before it has been subjected to the action of the gastric juice; though it is interesting to remember that it does not occur in those cases where the stomach is practically obliterated by a gastro-enterostomy.

'Bilious' diarrhœa, with its green motions, is supposed to be the result of a largely increased amount of bile entering the duodenum; it is more likely to be the result of an exalted peristalsis, commencing close to the stomach, which sweeps the liquid rapidly towards the anus. Unless peristalsis be also increased in the colon, diarrhœa cannot be said to be caused by it.

The diarrhœa which follows sudden chilling of the surface of the body may be caused by some interference with the normal microbes, or by such temporary vascular changes as prevent the absorption of the enormous quantities of succus entericus continually being poured into the canal in health, or it may fall under the following category.

'Eliminatory' diarrhœa is seen in Bright's disease, suppurative conditions, especially those of streptococcic origin, gout, &c., in which a poison circulating in the blood, upon being eliminated by the intestinal surface, acts as a purgative.

The diarrhœas caused by simple, tubercular, typhoid, dysenteric, or malignant ulceration belong to a different group. The intestinal contents, passing over the exposed nerve-endings in the ulcerated surface, cause greatly increased reflex peristalsis, which hurries them on, so that painful liquid motions occur. The condition is aggravated by the presence of inflammatory or catarrhal changes in the neighbourhood of the ulceration. Such fluxes are sometimes referred to as 'secondary' diarrhœas.

Pathologically, we may therefore consider diarrhœa as the manifestation or result of the action of a large number of complex organic chemical substances which possess cathartic properties. Clinically, however, all authorities use the term as practically synonymous with enteritis, of which numerous subdivisions or varieties are recognised, as jejunitis, typhlitis, colitis, entero-colitis, &c., according to the region involved, and usually with the prefix 'catarrhal.' In the opinion of the writer, it seems much more rational to regard the changes in the membrane which suggest these titles as simply secondary, being the result, and not the cause, of the condition.

In addition to the frequency and fluidity of the motions, which are the main characteristics of diarrhœa, pain and feverishness are also often present, both varying considerably in degree. The pain closely resembles that experienced

before the action of a brisk vegetable purgative, like senna. There are some nausea or even vomiting, loss of appetite, tympanitis, and thirst and dryness of the tongue in those cases, associated with fever. The number of the evacuations and the severity of the pain may roughly be accepted as a measure of the dose of the irritant swallowed in the food or manufactured in the bowel. The nature and intensity of the poison are evidenced in some ptomaine-cases, and notably in the variety of diarrhœa or enteritis known as cholera infantum, which is usually fatal, owing to the rapid absorption of the toxins.

Diarrhœa may become chronic after starting as an acute affection, but the majority of attacks last only a few days (rarely a week), recovery, as a rule, rapidly following the expulsion of the irritant from the bowel. The chronic cases, when not of nervous or ulcerative origin, may be caused by inflammatory or catarrhal conditions, which survive the expulsion of the irritants which originally induced them. Often they are the result of hepatic or valvular diseases.

Diagnosis.—In the great majority of cases this will simply resolve itself into the recognition of the cause of the catharsis, and often an examination of the stools and an investigation of the food swallowed will reveal the true nature of the case. Toxins in cheese, shell-fish, pork, mushrooms, canned meats, stale fish, milk, or beer are a common cause. The serious possibility of a mineral poison feloniously administered must not be forgotten in cases where no organic irritant can be traced. Diarrhœa induced by the presence of scybalous masses impacted in the colon, and the irritation caused by cancer of the rectum, should be carefully distinguished, as should also be the tenesmus occasioned by an intussusception.

The diagnosis of the site of the diarrhœa is often difficult. If the irritant act only upon the small intestine, much colicky pain and distress may be present without any diarrhœa ; irritation of the duodenum may cause jaundice and vomiting, and the motions will probably show the lienteric character and be bile-stained and devoid of mucus. When the irritant exerts its specific action upon the large bowel the pain may be intense, but sometimes it is absent ; probably in the former case the diarrhœa is the result of increased and irregular peristalsis : in the latter it may be due to simple increased secretion. There is much mucus generally present, and, when the rectum is irritated, great tenesmus. (See also the symptoms more fully described under 'Colitis.')

Though a markedly fetid odour may characterise diarrhœa from any acute microbic infection of the bowel, nevertheless, the eliminatory diarrhœa of septic or pyæmic conditions is often so indescribably horrid as to be distinctive.

In tubercular ulceration, the constitutional symptoms and the local signs of the abdominal glandular deposits are characteristic; but the marked feature of a painful diarrhœa alternating with bouts of constipation is very significant of tubercular mischief.

Prognosis.—This need hardly be discussed, as it will entirely depend upon the nature of the irritant causing the purgation. In the very young, and in the old and feeble, the gravity of the condition is of course increased. The forecast of the ulcerative cases is that of the primary disease.

Treatment.—If an acute attack of diarrhœa be considered as the result of the cathartic action of some chemical substance irritating the canal, the treatment is obvious. In the great majority of cases the agent expels itself, and it is wrong to interfere except with the view of relieving some urgent symptom, as very severe pain, or vomiting. Nothing can be more irrational than the dosage by large quantities of tannates which diminish secretion, and by opiates which paralyse peristalsis; both results intensify the mischief in the early stages. These may be indicated at a later stage, when the irritant has left behind it a catarrhal inflammation. A little judicious starvation, or milk-diet, with moderate rest, meets the requirements in most cases, allowing nature to effect her own cure.

Sometimes it may be necessary to assist nature by the administration of a purgative medicine, and there can hardly be a doubt about the drug which should be selected—viz. castor oil. This substance passes unchanged through the stomach, and the alkaline secretions in the duodenum set free its ricinolic acid, which expels the irritant, and at the same time the other constituents of the oil soothe the irritated mucous surface; one or two teaspoonfuls with a small dose of laudanum are generally enough. Rhubarb had better be withheld owing to its tendency to cause subsequent constipation, which is a thing to be avoided.

When the diarrhœa continues for two or three days, bismuth, chalk, and small doses of opium are indicated with the view of soothing or shielding the inflamed membrane from the alimentary contents as they pass towards the rectum. Tannin, especially in the form of tannalbin, may be given when there is evidence that the catarrhal condition of the membrane is leading to a greatly increased quantity of secretion. It is at this period that a dose of the dec. aloes co. gives such splendid results, its administration being often followed by the passage of a large semi-solid natural motion, after many days of a watery diarrhœa. Where rectal irritation is a prominent symptom, a small starch-and-laudanum enema or a morphia suppository should be given. Carminatives like cinnamon, clove, ginger, &c., often allay the griping pains by checking

irregularity in the peristalsis, and by their use opium may sometimes be dispensed with; peppermint-oil is valuable in this way.

When the diarrhœa is obviously caused by microbic action the ideal treatment would be an intestinal antiseptic, but all substances of this class are very unreliable. Salol and the naphthalin compounds are recommended, but they are inferior to calomel, which may be given in doses of a quarter of a grain every hour for six to ten doses. Brunton's plan of so managing the food as to cause the destruction or check the growth of the microbes is the best. He recommends that the milk-diet should be suddenly changed to a farinaceous one, which in a day or two is again replaced by a pure meat-juice diet, thus alternately eliminating the microbes which only flourish on these particular foods.

Where the diarrhœa is confined to the colon, large enemata of a weak antiseptic like permanganate of potash, borax, or saline solution may be employed to thoroughly irrigate the bowel, as described under 'Colitis.'

Nervous diarrhœa must be dealt with by removing as far as possible the mental causes; and it may be necessary to prohibit the patient for a time from placing himself in any position or surrounding in which an evacuation of the bowel would be highly inconvenient or impossible, as this is liable to bring on an attack. He should be told to refrain from going to stool as long as possible. One-eighth gr. morphia and 15 grs. tannalbin will often permit such patients to attend church or theatre; whether the powder acts by checking peristalsis and secretion, or by giving confidence to the nervous victim, is doubtful, but it often succeeds in the writer's hands. Morning diarrhœa may be sometimes prevented by similar treatment, and by abstention from all liquids after an early afternoon meal. For the diarrhœa which follows immediately after partaking of food, the best treatment is 30 grs. bismuth carbonate suspended in thin mucilage of acacia with 5 mins. morphia-liquor and 1 min. Fowler's solution, to be taken ten minutes before meals.

DIARRHŒA IN CHILDREN

This condition, generally described under the title of Enteritis of Infants, differs in no respect, pathologically, from the diarrhœa of adult life, save in the degree of its severity, and in its liability to end in a fatal issue, owing to the undeveloped resisting powers of the sufferer.

Etiology and Pathology.—So-called enteritis or diarrhœa is extremely rare in breast-fed infants. Its etiology is intimately associated with (1) bottle-feeding by cow's milk; (2) high summer temperature; (3) chills.

All the varieties of this condition are probably the result of microbic action. Several micro-organisms, notably *B. coli communis* and *B. aerogenes*, are always found in the intestines of healthy children, and it is possible that the former may take on virulent action under certain conditions which may lead to serious bowel-irritation. It is not, however, necessary to theorise upon this alteration in the activity of the B. coli, since numerous other organisms are constantly found in all cases of infantile diarrhœa. Booker has recognised as many as forty different varieties of microbe in infantile diarrhœa, all of which probably find their entrance through the milk of the cow ; and there need be no difficulty in realising the liability of the milk to infection, when one considers that by passing the sterilised hand over the skin of a milch cow and shaking the fingers above a gelatine plate myriads of micro-organisms can be recognised. Nash affirms that the house-fly is the carrier of the infection which produces epidemic infantile diarrhœa.

The microbes probably play no direct part in the irritation of the intestinal membrane, the cathartic elements being the virulent toxins which they manufacture from the proteids of the milk. Doubtless, the action of unorganised ferments must also be regarded as a cause in some cases, probably in the milder varieties of diarrhœa. The pathological anatomy of infantile diarrhœa hardly warrants the usual title of ' Enteritis ' applied to the disorder, since in many cases little change in the intestine may be observed, and such changes must be regarded as *secondary* and not of primary origin.

Redness and swelling, with some enlargement of the follicles, are often noticed, and ulceration in chronic cases is met with. These changes are obviously the result of the irritation or inflammation caused by the irritating chemical compounds elaborated by the bacteria, bacilli or cocci, and these organisms have been found in the lung and cerebral sinuses in cases complicated by broncho-pneumonia and thrombosis. Other chemical compounds resulting from the microbic action are absorbed, and produce the constitutional symptoms, or the specific cathartic substance itself may become absorbed and account for the intoxication.

Several distinct varieties of infantile diarrhœa are described. One common form is that in which the casein of the cow's milk is not digested, but, passing down in the form of firm masses of curd, is expelled with much pain, griping, tenesmus, and tympanitic distension. The attack may set in suddenly, with vomiting, fever, or a convulsion, and the motions are characteristic, consisting of greenish semi-liquid matter, with mucus and masses of whitish curd, which can be easily shaken off the moistened napkin. A similar condition may result in older children from eating unripe fruits and nuts ; in them the symptoms often cease at once after the

expulsion of the irritant. In infants the attack may be at once cut short on putting the child to the breast of a healthy nurse, or on suspending the feeding by cow's milk. This form is described by Osler as acute dyspeptic diarrhœa, and by Eustace Smith as simple non-inflammatory diarrhœa; it is common in all seasons, and may end fatally when not promptly treated by judicious feeding.

A more severe form common in cities, and in hot weather, and generally recognised as Acute Entero-colitis, or Inflammatory Diarrhœa, is met with frequently. Though it is most common in hot weather, it may occur at any season, and is not to be confounded with *Cholera infantum*, which is also known as *summer diarrhœa*, and which is much more frequently fatal.

It is certainly a microbic disease, or a condition the result of food-fermentation, and often the first symptom is that of gastric irritation. As its name implies, there is irritation ending in inflammation of the small and large intestine, both these, but especially the latter, often become the seat of ulceration, so that in protracted cases the symptoms take on a dysenteric character (see under 'Colitis'). The condition is also described as a follicular enteritis, and the inflammatory and ulcerative changes are the result of the injuries sustained by the mucous membrane through the virulence of the chemical substances resulting from the microbic or fermentative action in the bowel-contents, but the ulceration may be present long after the expulsion of the poisons has been complete.

Symptoms.—These set in with smart pains, followed by diarrhœa, which soon expels the semi-digested and fermenting food; after a short time the stools become liquid, offensive, and greenish in colour, and may be passed as often as every hour. There is smart fever with serious constitutional collapse; the pulse becomes quick and weak, the face pinched, the extremities cold, the skin dry, inelastic, and wrinkled; the fontanelles become depressed; vomiting may be as incessant as the diarrhœa, and the urine may be suppressed. The condition in severe cases, in forty-eight or seventy-two hours, may pass into the so-called hydrocephaloid state, and death from coma or collapse may result. As a rule, however, the progress is not so rapid, and many cases get well in about a week, whilst others may linger for three or four weeks, ending in recovery or death from slow exhaustion.

When the poison spends its force upon the large bowel the pain and tenesmus are very marked and distressing. The fluid motions contain much visible mucus and blood; the temperature runs high, and the collapse is sudden and often fatal, the clinical picture being such as might be expected from a typical acute colitis. As stated, the disease is most frequently met with in bottle-fed infants; but children may be attacked at

any age, and it may come on as a complication of other diseases, as the exanthemata, or it may be a sequel of the simpler non-inflammatory diarrhœa. The prognosis is better after the expiration of the years of infancy. It deepens, as a rule, with the height of the temperature and the frequency of the vomiting.

Treatment.—This should be conducted upon the same lines as those indicated in the simple non-inflammatory diarrhœa. Preventive measures are of first importance, and chief amongst these is the vital one of the purity of the milk-supply and the absolute cleanliness of the feeding-bottles and warming utensils. Sterilisation in very hot weather should always be resorted to, and the food prepared fresh every time before administration. The difficulty in many cases is that of supplying a substitute for cow's milk where breast-feeding is impossible. Substitutes for this, and the details of drug and dietetic treatment, may be found in the author's 'Dictionary of Treatment' (4th ed.), pp. 208–214.

Cholera infantum has already been briefly described upon page 208. It is an extremely fatal form of diarrhœa peculiar to cities, and occurs in bottle-fed infants, setting in with obstinate vomiting, intense thirst, frequent and copious watery stools devoid of fæcal materials, rapid shrinking of the skin and subcutaneous tissues, high fever and alarming collapse, ending often in the hydrocephaloid state, which rapidly passes into coma, convulsions, and death. The fatal termination may occur within twelve hours of the onset ; but if life be maintained for four or five days, recovery may be expected. It is evidently of the same pathological nature as the ordinary inflammatory diarrhœa and as English cholera ; the toxic microbic products are rapidly absorbed, and hence the predominance of the constitutional symptoms which produce a clinical picture of a severe intoxication.

The diarrhœa so frequently observed during the dentition period is probably of nervous or reflex origin, and is of little importance save when complicated or intensified by the supervention of the other varieties.

DIPHTHERIA

This is an acute infectious disease, generally involving the mucous membrane of the upper air-passages, caused by the presence of the Klebs-Loeffler bacillus, which, exciting a characteristic inflammatory action, produces a false membranous deposit from which the toxins of the bacillus are absorbed, causing neuritis and other constitutional disturbances.

The disease was recognised in the East in the second century, and has appeared in epidemic form at irregular intervals since

in all parts of the globe, and has now, with increasing prevalence, become endemic in most of the thickly populated districts of the earth, constantly breaking out in severe epidemics in cities and rural districts during the last half-century in Great Britain, Europe, and America. Bretonneau, first recognising its specific characters, gave to it the name Diphtheritis, which he afterwards changed to Diphtheria.

Etiology.—The disease spreads from one individual to another by personal contact, as has been frequently demonstrated by the outbreaks traced to schools and by the numerous instances of the infection of nurses and physicians. The bacillus can certainly be carried by persons who exhibit no symptoms of the disease, and it has been demonstrated to be capable of preserving its vitality for long periods in the clothing and about the crevices of apartments in which diphtheritic patients have been lying. It is probably never conveyed in the breath, but the fine spray diffused in coughing may be laden with the bacilli ; kissing is a constant source of the spread of the disease. Innumerable examples of its conveyance by means of the *milk-supply* are recorded, as it thrives in this medium, into which it probably gets an accidental introduction ; but Power believes that the milk may be contaminated owing to diphtheritic disease in the udder of the cow. The affection is also occasionally conveyed by cats suffering from it, and the writer knew of a case in which it was conveyed by the bite of a donkey ; and it is probable that it may be produced by a diseased condition affecting the throats of poultry, especially turkeys. Conveyance by means of the water-supply is considered not proven, and *water-borne* diphtheria is denied by most authorities. The same must be said of sewage and sewer-gas, and it is a significant fact that though the prevalence of enteric fever has been greatly diminished by improved sanitation, that of diphtheria has steadily increased of late years.

Newsholme maintains that the bacillus exists in the soil, and that the low level of the ground-water is the factor in epidemics, since these never occur in wet years, but in dry seasons following an unusually heavy rainfall, or during a season of rain which immediately follows a series of dry seasons.

The disease is most prevalent in the autumn; and though no age is exempt, more than three-fourths of the cases occur between the first and fifteenth years of life. Very young infants seldom take the disease ; girls and women suffer more frequently than males ; and the mortality is highest between the ages of infancy and the end of the fifth year.

Diphtheria is often associated with the members of the exanthemata, especially with scarlatina and measles. The bacillus may remain in the tonsils for many months after apparent convalescence, and it has often been found in the

throats of persons who have never shown any symptoms of the disease ; whilst some cases presenting the usual diphtheritic signs are found to be free from the bacillus.

Pathology and Morbid Anatomy.—There cannot be a doubt that the Klebs-Loeffler bacillus is the cause of the disease, but that this organism is capable of great variability in its virulence, as is evidenced by the remarkable differences in epidemics, is equally certain.

It is a rod-shaped non-motile bacillus, the shorter forms measuring about 3 μ, being about the length of the tubercular organism, whilst some of the longer rods may measure double this ; they are often clubbed at one end, bent somewhat, and apparently beaded, owing to the irregular way in which they take the stain. The bacilli are thus polymorphous, and vary greatly with the medium and method of culture, and show a marked tendency to group in clusters. They are found growing thickly upon the surface and in the upper layers of the false membrane, and more sparsely in the deeper layers, even to the depth of the mucous surface ; occasionally they have been discovered in the blood, lungs, spleen, and other organs, though this constitutional infection by the bacilli is no essential part of the disease.

The false membrane usually presents a whitish laminated appearance ; it is hyaline or fibrillated under the microscope, and, in addition to the Klebs-Loeffler bacillus, streptococci, pneumococci, and staphylococci are usually present in numbers. The membrane tears off with difficulty, leaving a bleeding surface. Another bacillus is commonly observed in the false membrane, and in cultures made from diphtheritic throats it is shorter and wedge-shaped, presents no beading, and does not vary its form by changing the culture-medium ; it is known as the pseudo-diphtheritic bacillus of Hoffmann, and is suspected of being an attenuated variety of the true bacillus.

The subject of the bacteriology of diphtheria is still in a confused state, owing to the remarkable variations in the staining and in the morphology of the bacillus both in the human throat and in cultures, and also owing to the fact that apparently identical bacilli are often found in the throats or nasal passages of perfectly healthy individuals. It is claimed that the specific Klebs-Loeffler bacillus always produces an acid reaction in glucose broth, and some bacteriologists refuse to accept all similar organisms as diphtheritic which do not yield this reaction, though they stain in a similar manner to the specific microbe. The intricacy of the subject may be appreciated by a consideration of Wesbrook's classification of the polymorphous varieties of the diphtheria bacillus into the three main groups of *granular*, *barred*, and *solid* or *even-staining*. No less than nine distinct types are thus recognised in this classification, as the *long* bacilli which are either granular,

barred, or solid-staining, and the *short* bacilli which may also be granular, barred, or solid-staining, and also the intermediate sizes. He considers that Hoffmann's pseudo-diphtheritic bacillus is probably the solid-staining variety of the short Klebs-Loeffler bacillus. The bacillus xerosis of other bacteriologists appears to be identical morphologically with the specific organism ; but it does not give the acid reaction in neutral broth, and it does not show virulence when animals are inoculated with it.

The subject is still further complicated by the fact that a *diphtheroid* inflammation is sometimes met with in the upper air-passages, which eventuates in the formation of a false membrane in which only streptococci may be discovered.

The false membrane varies in thickness and consistence ; thus sometimes, on the tonsils, it may be as firm as leather, whilst in the larynx and trachea it is soft and easily removable. There is always necrosis of the superficial layer of the mucous surface with its epithelium, and from the deeper layer comes the fibrinous exudation and leucocytes, which go to make up the false membrane ; in severe cases there is complete necrosis of the mucosa, which thus blends with and becomes part of the so-called false membrane. The membrane may be found over the tonsils, soft palate, fauces, nares, naso-pharynx, larynx, trachea, bronchi, conjunctiva, œsophagus, and even the stomach ; it is also found on superficial sores or wounded surfaces, and on the genital mucous membrane.

The lungs are often the seat of broncho-pneumonia caused by the presence of the bacilli. The lymphatic glands are swollen in the neighbourhood of the exudation, and often also in the mediastinum and in the abdomen ; the spleen and kidney are also enlarged and congested, and the solitary glands and Peyer's patches sometimes show considerable swelling. These changes are the result of the action of the toxins and not of the irritation produced by the bacilli. There is often distinct evidence of fatty degeneration of the cardiac muscle, which not infrequently leads to a fatal dilatation, and the bacilli have been found in vegetations on the valves. The most characteristic and constant change is to be met with in the nerves, as insisted upon by Sidney Martin. This consists of a rapid degeneration and breaking up of the white substance of the medullated fibres, and often rupture of the axis-cylinder. The nerve-lesions are peripheral, the central ganglia escaping ; the sensory and sympathetic fibres also suffer. Fatty degeneration of the muscle-fibres supplied by the affected nerves follows. That the Klebs-Loeffler bacillus is the direct cause of all the phenomena met with in diphtheria is practically demonstrated by experimental methods. It is easily obtained from the throat of the diphtheritic victim, and can be cultivated on suitable media. Pure cultures will produce the

membranous exudate in guinea-pigs, and the bacillus, when injected subcutaneously or into the veins, causes all the symptoms met with in diphtheria—viz. the characteristic degeneration of the nerves, heart, and muscles, broncho-pneumonia, &c., and from the blood and organs the toxins of the bacillus are obtainable.

When grown in broth for a time, the toxin or poisons which the bacillus secretes can be separated from the living virus, and the sterile chemical solution upon being injected is found to produce the characteristic or pathognomonic changes in the nerve-trunks always seen in fatal cases of human diphtheria.

It may therefore be accepted as proven that the Klebs-Loeffler bacillus as it grows in the false membrane on the human fauces secretes the toxin which, after its absorption, produces the characteristic constitutional symptoms. Sidney Martin believes that, though the bacilli are undoubtedly the cause of the diphtheritic poisoning, they accomplish this by the intervention of an unorganised ferment which they manufacture, which ferment has the power of splitting up the proteids into toxic albumoses. Whether this be correct or not, it does not interfere with the accepted view that the infection is a local one—*i.e.* one due to the absorption of the toxins locally elaborated at the site of the false membrane ; nor need this view be materially altered by the consideration of the fact that in some fatal cases the bacillus itself may find its way into the blood and internal organs. The result of the anti-toxin treatment itself is also conclusive evidence of the disease being produced by this specific bacillus.

It must not, however, be forgotten that the clinical picture of diphtheria varies very greatly, and this is a most important point in the consideration of its pathology ; as already noticed, other micro-organisms are often present, and these play an important *rôle* in the production of the symptoms in some cases. Most severe cases may be regarded as examples of a mixed infection, with staphylococci, streptococci, or pneumococci, these organisms explaining the marked tendency to suppurative lesions and the partial failure of the antitoxin treatment, unless when buttressed by injection of antistreptococcal serum.

Symptoms.—The period of incubation varies exceedingly. It may be as short as twenty-four hours or as long as five or six days ; forty-eight to seventy-two hours may be accepted as a common period.

The first symptoms are generally a slight febrile disturbance with malaise. If the child be old enough to express his sensations, some pain in the act of swallowing may be complained of before or soon after the pyrexia. There is tenderness and swelling in the lymphatic glands about the angle of the jaw, and if the throat be inspected at this early stage, amidst some

extra redness of the membrane covering the fauces, soft palate, or tonsils, a thin whitish patch may be noticed, at first almost pellucid, but soon becoming opaque and white or yellowish-white. There may be several such patches, which in a short time coalesce and spread over both tonsils and soft palate, so that, in a severe case, in forty-eight hours the entire upper part of the throat may be covered with a continuous thick creamy-white membrane. In cases inspected early, uniform or patchy redness may be noticed without any evidence of membrane ; but very generally, when first coming under observation, one or more characteristic white spots are obvious on the surface of the tonsil or on the pillars of the fauces, which are at first liable to be confounded with the yellowish or greenish-white pellets often met with in the tonsillar hollows.

Even at this early stage some idea of the gravity of the case may be obtained by watching the rapidity of the growth of the membrane, and, if a portion be detached by the forceps, a fresh growth may appear upon the bleeding inflamed surface within three or four hours. If the case be promptly treated by serum injection, the patch may cease to show signs of spreading, and rapid disintegration may follow, leaving a reddened base which may show slight signs of ulceration. In untreated severe cases the false membrane increases in thickness, and much local swelling or œdema occurs ; it becomes gradually darker in colour, decomposes, and forms a slough-like mass, giving out an offensive putrid odour, and accompanied often with purulent secretion ; and about the sixth or seventh day it is thrown off, but it may form again upon the same site if life be prolonged.

Sometimes the formation of false membrane is indistinct or absent, and, instead of the firm wash-leather exudate, the throat may be covered with a gelatinous or pultaceous film, or, as in latent diphtheria, only a diffuse dirty-redness may be observable.

As the local signs progress, in moderately severe cases, the swelling and tenderness of the lymphatic glands increase ; the fever continues, but it seldom exceeds 102° or 103°, though the pulse may remain much higher and show characters indicative of weakness.

Albuminuria is very commonly present, setting in as early as the third or fourth day, and, as it bears some proportion to the severity of the constitutional poisoning, its presence and amount should be carefully noted. It occurs in more than 50 per cent. of all cases, and exists in the great majority of the severe and fatal examples of the disease ; and in these *suppression* of urine sometimes follows.

Mild cases of diphtheria may show no symptoms indicative of constitutional infection till after the disappearance of the

local signs, and the writer has seen several such cases followed by marked paralysis, and not a few where serious paralytic symptoms have first directed attention to the history of a throat-affection which, owing to its insignificance, had been entirely overlooked or disregarded by the patient.

At the expiration of about a week, in moderately mild cases, the membrane has disappeared, and in a few days more convalescence sets in.

In severe cases all the symptoms deepen, the local manifestations take on a more active character, the membrane extends often into the nares or downwards towards the larynx, the swelling in the lymphatic glands becomes great and the tissues in the neck swollen, so that swallowing becomes most difficult or impossible, and even suppuration may occur, though this is a rare event. Free muco-purulent acrid secretion flows from the nostril, excoriating the lip; fœtor becomes evident, as sloughing of the false membrane occurs, and otitis media may appear. The constitutional symptoms deepen; the fever generally rises, though in fatal cases it usually falls to below normal; the pulse gets very frequent, weak, and uncountable; prostration becomes more marked, though consciousness is not interfered with; and cardiac failure becomes more evident. Unless broncho-pneumonia or suppression of urine occurs the patient gradually sinks from exhaustion of the heart-muscle caused by the diphtheritic toxins, and accelerated usually by constant vomiting. Heart-failure may, however, set in suddenly and prove rapidly fatal at any stage of the disease—*i.e.* as early as the fifth day, or during the paralytic stage, many weeks after all local signs have disappeared. It may show itself as the result of acute dilatation with mechanical embarrassment of the breathing and circulation. The false membrane may be limited to the fauces or pharynx all through the attack, or, as just mentioned, it may extend to the nostrils or larynx; but it may, upon the other hand, originate in and remain confined to the nostril, constituting *nasal* diphtheria, or it may restrict itself to the larynx as the true *laryngeal* affection, which will be described separately.

Complications and Sequelæ.—Some of these have been already mentioned, as suppuration of the lymphatic glands, broncho-pneumonia, otitis, and suppression of urine. The complications of a septic nature are supposed to have their origin in the infection produced by the microbes which often accompany the Klebs-Loeffler bacillus. These, in addition to the production of *abscesses* and *pyæmia*, are the probable cause of the *lobar pneumonia*, *otitis*, *endocarditis*, and *thrombosis* met with in the disease. The *broncho-pneumonia* may have the same origin, but undoubtedly it can be caused by the diphtheritic bacillus; and the same may be said of the

nephritis which occasionally follows; but the *albuminuria* met with so frequently in diphtheria should not be regarded as a complication: it is the result of the toxin circulating in the blood, and may be looked upon as a symptom of the disease present in a large proportion of cases. *Hæmorrhages* may occur from the necrotic action involving vessels in the region of ulceration; but a serious form of the disease (hæmorrhagic diphtheria) is occasionally met with, in which a purpuric condition of the blood leads to bleedings from the mucous membranes and into the skin and retina.

Urticaria, *erythema*, and *erysipelatous rashes* are often noticed; but these are the result of the serum-injections in some cases.

Paralysis, the result of a multiple neuritis, is by far the most important complication or sequela of diphtheria, though, strictly speaking, it is neither, but should be regarded as a symptom of the disease, since it is demonstrated to be the most constant of the results of intoxication by the diphtheritic toxins. It sometimes, though not often, appears when the throat-mischief is at its height—*i.e.* before the end of the first week from the onset of the symptoms; but more commonly it may be regarded as a sequela, appearing as it generally does in about two or three weeks after apparent convalescence. It is most frequent in faucial diphtheria and rare in the laryngeal form; and though the percentage of cases in which it supervenes varies in different epidemics, it may be roughly recognised as about one in seven or eight of all cases of pharyngeal diphtheria. It may follow the mildest cases and those in which the throat-symptoms have been overlooked; and this is perhaps the reason for the statement, which is certainly inaccurate, that it is more liable to supervene upon very mild forms of the disease.

The first symptoms of diphtheritic paralysis are usually produced by weakness of the soft palate, which causes the voice to become nasal in character, and permits of the regurgitation of fluids through the nostrils in the act of drinking. Upon inspection, the palate is seen to have lost its arching and to hang down loosely, its reflex, its sensibility, and its power of motion being destroyed upon one or both sides. The constrictors of the pharynx may suffer, so that swallowing becomes impossible or very difficult, the paralysis of the lower one permitting the entrance of food to the larynx. Often the condition extends no further than the palate, which may recover its power in two or three weeks; but nearly every group of muscles in the body may suffer. The eye-muscles are very commonly affected: loss of accommodation, through paralysis of the ciliary muscle, may be the first symptom; squint, ptosis, and double vision may also occur. Paralysis of the lower extremities is much more frequent than that of the arms;

but both arms and legs may be affected together, and wasting is commonly noticed. The muscles respond but feebly, or not at all, to faradisation; though the nerve-trunks show no change in their normal reactions, the paresis of the limbs goes on deepening for six to eight weeks before a paralytic state is reached. Gradual improvement, as a rule, follows, though it may take quite as many months before power is finally restored in slow cases.

The knee-jerks are lost early, and this may be the only sign of paralysis; the pupil-reflex is sluggish, but not abolished. Laryngeal paralysis causes aphonia, and danger of suffocation and aspiration-pneumonia may be produced by the entrance of food into the bronchial tubes. The facial muscles, tongue, gullet, and the sphincters generally escape, though the trunk-muscles, the intercostals, and diaphragm may suffer. There is often impaired sensation, and the weakened muscles may be painful. In cases where the paralysis is general, the patient lies absolutely helpless, though quite conscious, affording a clinical picture which cannot be mistaken.

The pathology of the paralysis is certainly that of a multiple neuritis produced by the action of the absorbed toxins. (See under 'Neuritis.')

The serious tendency to cardiac failure has already been referred to, and in the paralytic stage it may be caused by the neuritis of the heart-nerves, ending in heart-block.

Relapses in diphtheria are not common; but in those cases where there are chronically enlarged tonsils and adenoid growths, the Klebs-Loeffler bacillus may remain in the throat for many months, and repeated mild attacks may from time to time occur till surgical measures be resorted to with the view of clearing away all unhealthy tissue.

Diagnosis.—The presence of the specific bacillus must be held to be the final test in diagnosis, and in all doubtful cases it should be carefully searched for. It may be detected by an experienced microscopist on a direct examination of a swab from the affected throat, but more usually a culture is first made from a piece of the false membrane, or from the secretion wiped off from the membrane or suspected exudate by means of a sterilised cotton-wool swab rubbed firmly over its surface. The swab is smeared over the surface of some sterilised blood-serum in a test-tube, which is then placed in an incubator at 37° C. (98·6° F.) for twelve hours, when the bacillus may be recognised under the microscope. To the naked eye the colonies appear as an opaque white streak or in distinct small dots. In laryngeal cases the swab is pushed against the back of the pharynx or pillars of the fauces, and treated as before. When the incubation is permitted for a longer period other organisms make their appearance. In doubtful cases one negative result should not be taken as conclusive.

In unquestionable cases of diphtheria a visible membrane may never form, and it is quite untenable to maintain that these are not diphtheritic, though the bacillus is present, because experience has proven that such non-membranous cases may convey the typical disease to others, and, moreover, they may be followed by the characteristic paralysis later on. The subject of these latent forms of diphtheria has been exhaustively dealt with by Watson Williams in the 'Journal of Laryngology' for November 1905.

Hence *catarrhal sore-throat, simple* and *follicular tonsillitis*, and *ulcerative tonsillitis* can often only be excluded by a careful bacterial examination, or, in the absence of this, by repeated inspection of the suspected spots on the tonsils and pharynx, the patient being in the meantime strictly isolated. *Acute follicular tonsillitis* has generally a very abrupt onset, with constitutional symptoms as severe as those met with in 'grippe'; the backache is often as prominent as that met with in small-pox, and there is nearly always a history of repeated attacks. The punctate yellowish-white spots or plugs correspond to the distended crypts, which may exhibit an ulcerated appearance. The widespread or universal redness of an ordinary *catarrhal sore-throat*, with its free mucous secretion, is generally distinctive.

Vincent's angina is a disease produced by the presence of his fusiform bacillus, alone or in association with a spirillum or spirochæte. It may only attack the tonsils, or it may spread to the palate, and even appear on the gums or buccal membrane, and at first sight simulates diphtheria, especially as the adjoining lymphatic glands become early involved. The essential lesion in this throat-affection is not a true membranous formation, but a necrosis of the upper layers of the mucosa which causes a greenish-yellow fetid slough, which is liable to be mistaken for a diphtheritic membrane. The necrosis may extend deeply, and has been known to invade the larynx. The organisms stain readily with any aniline dye; the fusiform bacillus is always found in considerable masses; the spirochætes are comparatively few.

Caustics, especially nitrate of silver and strong carbolic acid, when applied in any form of sore-throat, may cause an appearance which is sometimes mistaken for diphtheria.

Herpes of the palate seldom occurs unless when associated with herpes of the lips, and its subsequent course negatives diphtheria.

Erysipelas of the throat and *quinsy* are excluded by their abrupt and severe onset with high fever often associated with rigors and accompanied by great swelling or œdema, and in the case of quinsy rapidly passing into pus-formation; when there is the appearance of a membrane in such cases it is caused by a pultaceous mucous deposit differing from the

typical false membrane in its softer consistence and transparency.

Ulcerative and *follicular stomatitis* are easily excluded by the marked tendency to ulceration of the mucous membrane of the gums, tongue, and cheeks; and *thrush* by the thin white film, which is easily wiped off and which exhibits under the microscope the branching filaments of its specific fungus.

Scarlatina in its early stages is sometimes mistaken for diphtheria when the rash is faint or has disappeared early, as the throat may show a pultaceous, mucoid, viscid exudate, which occasionally closely resembles the characteristic false membrane produced by the Klebs-Loeffler bacillus. The tendency of the scarlatinal throat to ulcerate deeply under the membranous exudation is characteristic, and the grave constitutional symptoms and fever, even in the absence of a rash, may often settle the diagnosis. The absence of the bacillus on bacteriological examination is conclusive. At a later stage of the scarlatina genuine diphtheria with its typical membrane may appear.

In *enteric fever*, in rare cases, at the onset a false membrane may be visible, but the bacteriological examination settles the doubt.

Syphilitic ulceration and mucous patches are recognised by the other evidence of syphilis.

The diagnosis of the laryngeal form will be mentioned in the description of membranous croup.

Prognosis.—The rate of mortality varies widely in different epidemics. It may be safely stated that it is considerably less than half what it was over the civilised world before the introduction of the antitoxin treatment; in some countries it has been reduced to one-fourth the former rate. In scarlatinal diphtheria treated at the onset the mortality has been reduced to one-tenth of the old rate. The younger the patient, as a rule, the more serious the forecast, and certainly the prognosis deepens with every hour which intervenes between the onset of the disease and the antitoxin injections. The serious nature of the various points which indicate danger has been dwelt upon in the description of the symptoms, such as the extent of the membrane, cardiac failure, suppression of urine, broncho-pneumonia, &c. Hæmorrhagic cases are especially fatal. Paralysis, if not fatal by interfering with the respiration and heart, is always recovered from. Frequent vomiting is a grave symptom; irregularity and marked weakness of the pulse, with coldness of the extremities and a fall of the temperature in the mouth or rectum, are signs of approaching death. The prognosis in cases where the membrane extends to the larynx is always grave.

Treatment. — As regards feeding, isolation, nursing, the administration of stimulants, and the treatment of such

symptoms as vomiting, fever, &c., these must be conducted according to the universally recognised principles.

Local measures directed to the destruction of the false membrane by caustics and powerful antiseptics are now practically abandoned. The throat should be sprayed continually by a weak and bland antiseptic solution from the beginning, and nothing is better than a 1 per cent. solution of carbolic acid, or a saturated solution of boric acid.

Antitoxic injection should be resorted to at the earliest possible period, and the age of the patient need not enter into the question of dosage. Where the patient is seen within twenty-four hours of the onset a dose of 1,500 to 2,000 units should be injected into the subcutaneous tissue of the abdomen or loin by a suitable syringe under strict antiseptic precautions. In mild cases such improvement follows in the appearance of the throat-condition in twenty-four hours that further injections may be unnecessary; if any doubt exists, a second injection of a similar amount should be given. Severe cases may upon the first day receive 2,000 units, which may be repeated in twelve hours. Where two or three days have supervened before treatment, 4,000 units should be given in all severe cases, and this dose may be repeated at the end of twelve hours. Later severe cases will require an initial dose of 8,000 units with repetitions of 2,000 units every twelve hours. Apparently hopeless cases have recovered after doses of 30,000 units, and no case should be abandoned, no matter how apparently hopeless, without administering the serum in liberal quantity. The only untoward effects observed after serum-injections are the erythematous rashes and arthritis, which need cause no anxiety; abscesses are rare, and nearly always preventible by careful antiseptic precautions.

Louis Cairns has published the results of twenty cases of very severe diphtheria treated by *intravenous injection* of 25,000 units, and it would appear that this method should get a trial in the malignant forms, and especially in the hæmorrhagic variety, where pulmonary complications exist; the dose should be repeated in twenty-four hours if necessary. Whether given hypodermically or by the veins, there is no limit to the amount of serum which should be used; the dosage can only be gauged by the symptoms which follow, and the remedy should be pushed till shrivelling or disintegration of the false membrane becomes evident, and a general improvement in all the symptoms sets in.

The *rationale* of the serum-therapy, and the experimental facts upon which it is based, are described in detail in the author's 'Dictionary of Treatment,' at pp. 214–223, together with a survey of the various local measures and internal remedies still recognised in the treatment of diphtheria. The

treatment of heart-failure must be prompt, and its advent always anticipated or watched for, the first signs of marked weakness being met by full doses of strychnine (*i.e.* $\frac{1}{15}$ to $\frac{1}{10}$ gr.) and a strict observance of the recumbent position.

Paralysis of limbs requires little special attention: gentle massage, faradisation alternated with the continuous current, and deep injections of small doses of strychnine, will usually expedite recovery, which in every case is certain; but these agents should not be resorted to in the early stage. When the constrictors of the pharynx and the larynx are involved in the paralysis, nasal feeding by the rubber tube should be commenced, and rectal feeding may be also resorted to. *Preventive* measures in times of epidemic are to be carried out by rigid isolation of those affected, as in the management of scarlatina and small-pox. The diphtheritic convalescent should be regarded as a possible source of infection for at least four weeks after the expiration of the active stage, and even then he should use the weak carbolic throat-spray, as the bacilli may be still found in the tonsils; and close personal contact, as in kissing and sleeping in the same bed with healthy children, should be forbidden for longer periods.

The antitoxic serum secures immunity to those in close contact with diphtheritic patients, but only for the brief period of two or three weeks; and injection of moderate doses —*i.e.* 1,000 units—may under exceptional circumstances be resorted to in schools and dormitories. It must always be remembered that even a severe attack of the disease does not confer long immunity from further attacks, as is the rule in the exanthemata.

DIPHTHERIA OF THE LARYNX

This is recognised by most authorities as a synonym of *True Croup* or *Membranous Croup*. In the majority of cases it begins in the fauces and extends downwards, when laryngeal symptoms supervene, after three, four, or five days, upon the ordinary signs and symptoms of diphtheria. In such cases there can be no difficulty in diagnosis or room for difference of opinion about its pathology, as the Klebs-Loeffler bacillus is probably always present.

Pathology.—*Primary* laryngeal diphtheria, or true membranous croup, beginning in the larynx, is a well-recognised affection; and owing to the narrowness of the upper portion of the air-passage in children the symptoms are more serious and obvious than in the case of the adult. In these cases the specific bacillus is often to be found in cultures made from a cotton-wool swab applied to the pharynx, although no visible exudate or membrane exists there.

The important question arises, whether all cases of membranous laryngitis, or true croup, should be considered as genuine cases of diphtheria caused by the specific bacillus. The writer is thoroughly convinced that this view, in the face of clinical experience, is not correct. In a considerable percentage of cases the diphtheritic bacillus is not to be found : according to Osler's figures, it was absent in about 20 per cent. ; but this cannot be accepted as a valid argument against the specific nature of the membranous inflammation of the larnyx, as the bacillus is sometimes not to be detected even in faucial cases where a characteristic membrane is visible to the naked eye, and where the constitutional symptoms of diphtheria exist also in a well-marked form. In districts where true faucial diphtheria is never, or very seldom, seen, cases showing all the clinical characters of true membranous croup occur often in children, and are generally fatal. Such cases are not infectious, and the few which recover do not develop any paralytic symptoms. Such was the writer's experience during a period of about ten years in an extensive general practice, when such cases were constantly being met with. The closest scrutiny failed to find any evidence of the disease being communicated to those coming into close personal contact with the sufferers, and during all this period no case of faucial or nasal diphtheria was known in the district. When afterwards an epidemic of diphtheria occurred, and later when this disease became endemic, he could not satisfy himself that the proportion of such cases occurring bècame obviously greater. The period referred to was before the discovery of the specific bacillus and the antitoxin-treatment.

Whilst from a purely clinical standpoint it would appear certain that all cases of true membranous croup are not diphtheria, there is, however, a difficulty in accepting such a distinction from the therapeutic standpoint. Undoubtedly the introduction of the serum-treatment has lowered the terrible mortality of these cases. This is perhaps most obvious in the examples of primary membranous croup where tracheotomy has to be undertaken ; for though the serum-treatment is often useless as a substitute for the operation, it certainly saves life when used in conjunction with it. In the pre-serum days recovery after tracheotomy in infants was rare in this country ; the writer was not able to record a single success in his own cases. If recovery follows frequently under serum-therapy in apparently hopeless cases of membranous croup, the assumption is that the disease is diphtheria.

Still, after allowing for such reduction in the mortality as is seen under serum-therapy, there remains a residue of cases inexplicable under any other hypothesis than that of there being a diphtheroid membranous croup or laryngitis, which is

not infectious, and which is not caused by the specific bacillus. Whether streptococcus or other organism is capable of exciting the membranous inflammation is not clearly proven.

Symptoms.—These consist, in the cases of patients already suffering from faucial diphtheria, of the gradual or sudden supervention of symptoms indicative of interference with the voice and breathing. In primary laryngeal diphtheria, or true membranous croup, whether diphtheritic or not, the symptoms are identical. The first thing to attract attention is slight malaise accompanied or soon followed by some degree of hoarseness in the voice, and this paraphonia, as it is called, is evidenced also in the sound produced on coughing. Generally the hoarseness, croupy cough, and malaise last for two or three days in such a mild degree as to attract little attention or anxiety—a state of affairs contrasting markedly with the sudden invasion witnessed in simple catarrhal laryngitis or spasmodic croup.

With more or less suddenness the symptoms deepen, and difficulty of breathing becomes evident, coming on in paroxysms of either inspiratory or expiratory dyspnœa, the first attack often being noticed in the middle of the night. Stridor, or a whistling noise produced by the current of air passing through the narrowed larynx or trachea, may be the first alarming symptom. When the dyspnœa becomes established the movements of the chest are obviously interfered with, all the inspiratory muscles act powerfully, and the yielding parts of the thorax and the epigastrium recede. The difficulty in some cases is most marked in expiration, which becomes unduly prolonged and increasingly laboured, and the nostrils dilate. The cough and voice get more husky, and aphonia may become complete. During the first twenty to forty hours, in the intervals between the attacks of dyspnœa caused by laryngeal spasm, the symptoms brighten, but when the difficulty of breathing is sufficient to show marked recession of the lower costal spaces there are no intervals of relief, the dyspnœa having become continuous from laryngeal obstruction caused by the membranous exudate.

With the obstruction of the larynx, the blood is imperfectly aërated; lividity of the lips, ears, and finger-tips, deepening to cyanosis, is seen; great restlessness, quickness and weakness of the pulse become evident; and the child gradually sinks from suffocation unless in some violent expiratory effort the membrane is coughed up and expelled. This is, however, more likely to happen if the seat of the exudation be tracheal or bronchial. Fever is generally slight and often absent. In adults there may be aphonia without any signs of laryngeal obstruction, owing to the greater width of the glottis; continuous dyspnœa in these cases may mean a blocking of the

trachea and bronchi, which is relieved only when casts of the lower air-passages are expelled by coughing or vomiting.

Tracheotomy may be urgently indicated at any period by the sudden supervention of symptoms of suffocation; and the writer has performed the operation with satisfactory result even after breathing had entirely ceased.

Occasionally in primary laryngeal cases the growth of the membrane may extend upwards, and the specific nature of the disease may be visible by the development of typical patches of diphtheritic membrane on the tonsils or fauces, in which cases the glands at the angle of the jaw enlarge, and fever becomes marked, with other symptoms of constitutional infection which are rare or absent in most cases of primary membranous croup. Albuminuria is often absent, and paralysis seldom follows where the mischief is confined from the first to the larynx.

Diagnosis.—There is usually not much difficulty in locating the disease, as from the foregoing description it is obvious that laryngeal obstruction is the prominent feature from the beginning. The laryngoscope is generally of no use for demonstrating the presence of membrane in children, but by forcibly depressing the base of the tongue the epiglottis may be easily seen in most cases without the aid of the throat-mirror, and sometimes the false membrane can be viewed on its tip.

Catarrhal laryngitis, false or *spasmodic croup*, has a very sudden onset, and is never fatal, though the stridor and dyspnœa may be very prominent features for an hour or two; the voice and cough may be hoarse, but aphonia does not occur.

Post-pharyngeal abscesses, adenoids, abscess or *enlargement of the tonsils* can usually be eliminated by a careful examination of the throat by the eye and finger-tip.

Scalds of the larynx causing *œdema* of the glottis from inhaling the steam of a kettle simulate true croup, but the extent of the œdema and the history of the case, when obtainable, settle the diagnosis.

Foreign bodies, as coins, beads, &c., sucked into the larynx by young children who can give no history of the accident may cause serious difficulty in diagnosis; the sudden explosive nature of the onset, resembling laryngismus stridulus, should arouse suspicion. If the initial spasm does not prove fatal, the laryngoscope or the X-rays may demonstrate the presence of the foreign body.

Measles before the appearance of the rash may present acute laryngeal symptoms and croupy cough, caused by spasm supervening upon the laryngeal catarrh, but the presence of high fever, coryza, lachrymation, and suffusion of the conjunctiva, will settle the diagnosis. Laryngeal obstruction coming on at a later period after the disappearance of the

rash, as in the case of *scarlatina*, may be caused by a true diphtheritic membrane.

Paralysis of the abductors of the vocal cords and *syphilitic* or *tubercular ulceration* only occur in adults, and are diagnosed by the aid of the laryngeal mirror.

Prognosis.—This is always very grave, and gravest in very young children. In the primary laryngeal form in adults the prognosis is very grave where evidence of membrane exists in the bronchial tubes or trachea.

Treatment.—As soon as laryngeal symptoms demonstrate the presence of membranous formation a brisk emetic should be administered. The child should be placed in a cot, or upon the knee of a skilled nurse inside a canopy of curtains in which a moist warm atmosphere can be continuously maintained by the bronchitis-kettle. A dose of antitoxic serum (2,000 units) should be injected without delay and repeated in twelve hours, or sooner, if the symptoms be not relieved.

Tracheotomy or intubation in the majority of cases will be indicated. (The steps of both operations are given in detail, along with the after-treatment and local measures, in the author's 'Dictionary of Treatment,' 4th ed., pp. 180–190.) After tracheotomy the injections should be maintained, and apparently hopeless cases may only show signs of improvement when the dosage has reached a total of 50,000 units.

DIPSOMANIA

This is a paroxysmal recurrent form of insanity, manifesting itself in periodical attacks of alcoholism. It is not a habit-disease, and should not be confused with chronic alcoholism. There is often a history of insanity or of some neurosis which aids one in understanding that the dipsomaniac is born with an idiosyncrasy towards alcohol, and that, after discovering the effect which it produces upon him he is wholly unable to resist the temptation of getting drunk. After the first debauch the desire for alcohol passes away for a longer or shorter period ; it may be several months or years before the craving returns, during which the victim lives a sober, regular life. Restlessness, insomnia, mental depression, and irritability mark the return of the craving, which may be resisted to the extent of producing intense misery ; but generally, though the earlier attacks may be successfully warded off, the common result is that the dipsomaniac yields and plunges without restraint into a drinking bout in which he may be more or less continuously drunk for many days. Recovery from his debauch is usually associated with deep humiliation of spirit, generally with a repugnance to alcohol. The intervals

between the attacks are variable, though in some cases a regular periodicity is observed.

When the attacks occur frequently, with short intervals of sobriety and prolonged periods of indulgence, the disease tends to pass into almost continuous drunkenness, and the alcoholic habit becomes grafted upon the original dipsomania. Delirium tremens may set in during or at the end of one of the fits of drinking. In rare cases one deep alcoholic draught may completely satisfy the craving without drunkenness supervening.

Treatment.—The underlying inherited or congenital mental peculiarity or idiosyncrasy is beyond the reach of treatment, but by discipline and moral training the victim of this disease may be sometimes taught how to fight, and be aided to such an extent that he may overcome, the acute pressure of temptation. When the premonitory signs warn him of an impending attack he should be helped to take such prompt steps as shall render it impossible for him to procure drink ; thus some patients seek an asylum or retreat when they feel the early restlessness and depression coming on, whilst others seek safety in a total change of their environment.

Hypnotism, when skilfully employed, has yielded excellent results in some patients. The treatment of the patient when indulging and when recovering from his bout of drunkenness will be that described under 'Alcoholism' and 'Delirium Tremens.'

DISSEMINATED SCLEROSIS

This, also known as Insular or Multiple Sclerosis, is a chronic disease of the brain and spinal cord, characterised by the formation of scattered plaques or islets of sclerosis caused by an overgrowth of the neuroglia which takes the place of the nerve-elements. The brain or spinal cord may alone be affected in rare atypical cases, but the cerebro-spinal form is that which is generally met with and recognised clinically.

Etiology.—Numerous causes have been assigned, but the etiology is still unsolved. Infectious fevers, especially scarlatina, poisoning by lead, exposure to cold, trauma, heredity, prolonged anxiety, and many other conditions, have been supposed to act as exciting or predisposing causes. It affects both sexes about equally, and most frequently commences between the ages of twenty and thirty ; children are sometimes affected, but the disease is very rare in advanced age.

Pathological Anatomy.—The sclerotic patches are found scattered irregularly throughout the brain and cord, and, though much more numerous in the white, the grey matter does not escape, especially that of the basal ganglia ; the

cranial nerves and spinal roots also suffer. The patches are of a reddish-grey colour, irregular in outline, and sharply defined to the naked eye. To the finger they may feel soft, but in old cases they are of firm consistence ; their size varies from that of a hemp-seed to that of a large bean. On section they do not project above the surface, though when studded over the surfaces of the pons, crus, and cord they may seem to project slightly. They may invade the cord throughout its entire thickness as they are seen to do in the brain, but they are not generally visible on the surface of the grey matter of the hemispheres.

Under the microscope the islets are found to be the result of marked proliferation of the interstitial tissue ; the nerves are deprived of their myelin sheaths, the axis-cylinders are preserved and also the ganglion-cells ; hence there is no secondary degeneration of the fibres except in the immediate neighbourhood of the sclerosed patch.

Pathology.—The disease is probably a primary interstitial inflammation which leads to secondary destruction of the myelin sheaths ; though some maintain that the parenchymatous change is primary, the proliferation in the neuroglia being secondary. It is probable that the starting-point is a toxin produced by some bacillary disease, which may have attacked he patient long before and been overlooked or forgotten. Charcot believed that the tremors, so characteristic of the affection, were caused by retardation of the impulses passing down the denuded axis-cylinders ; whilst Erb considers they are the result of derangement of the coördinating function produced by the plaques.

Symptoms.—The four prominent symptoms of the disease when established are the *tremors*, *muscular weakness*, *scanning speech*, and *nystagmus*. The onset is gradual, the progress slow, and the termination invariably death. The clinical picture varies greatly, and though the disease must be considered as essentially progressive, it is very often marked by periods of arrest so apparently complete as to raise the hope of an error in diagnosis. This is almost a distinctive character in the atypical cases which resemble hysteria, and in them the initial symptoms of loss of power in one or more limbs, numbness, impairment of sight, &c., may entirely disappear for many months or years, to return again in conjunction with the classic symptoms and signs later on. Hence there is considerable difficulty in any attempt to divide the progress of the disease into stages.

The first symptom is often a weakness in the lower extremities or some symptoms of incoördination, upon which gradually supervene spasticity, increased tendon-reflex, Babinski's sign, ankle-clonus, and ultimately, in the final stage, contracture. The muscles are not at first atrophied, and

their electrical excitability little, if at all, changed. The condition will at this stage be that of a spastic paraplegia, which later on compels the patient to remain permanently in bed, perhaps doomed to catheter-life. There may not be for many months or even years an appearance of the other typical symptoms—a fact which shows the great variation or polymorphous character of the clinical phenomena. But in the marked cases, with this spastic paresis of the lower extremities, soon there appears the characteristic tremor in the arms and hands, though these may show little evidence of motor weakness.

The *tremor* is described as a volitional or intention-tremor; *it is not visible when the muscles are at rest*, but is brought into action by the patient attempting to grasp any object, when a series of jerking movements are executed which frustrate his aim and which soon render him unable to use his hands or feed himself. The tremor may spread to the head and neck, and the legs may be affected.

Nystagmus may be regarded as evidence of this tremor affecting the eye-muscles, though it sometimes may there be visible as a series of spontaneous jerky movements of the eyeballs. It is, however, always much intensified on bringing the globe-muscles into action, and in the early stages may be only evident upon turning the eyeballs outwards. Like the tremor, nystagmus is present in nearly three out of every four typical cases.

Scanning speech is not so frequently present, but it is a common and equally characteristic symptom. The words are uttered slowly in a staccato manner, each syllable being separated and accentuated ; the symptom is best marked after the muscles employed in speaking become tired. True aphasia is very seldom met with.

Optic-disc changes and defects of vision occur in nearly half the cases, the characteristic appearance being marked pallor and atrophy, though optic *neuritis* is rare, and amblyopia may be prominent without the disc appearing abnormal. Sensory disturbances, as numbness and anæsthesia, are not uncommon. Severe headache and vertigo or giddiness may be early symptoms when the cerebrum at first is mainly involved ; mental weakness, as shown by emotional excitability and delusions, and at a later stage by a tendency towards dementia, may be evident. The final physiognomy of the disease is often somewhat characteristic : there is a stolid, vacant, or heavy dull expression, with tremors of the lips or facial muscles, when speaking is attempted.

The sphincters are not much interfered with, as a rule, until the disease enters upon its late stages ; and trophic disturbances belong to the same period. Epileptiform convulsions, apoplectiform or hemiplegic attacks ending in a transient

coma may appear at any stage, or may be the termination of the disease. Bulbar symptoms are not rare.

It is impossible to forecast the duration of the disease, the cases vary so widely owing to the extent and localisation of the plaques; thus the same cause which produces such variations in the symptoms affects also the duration of the affection. The disease is an essentially chronic one, and patients may live in a bed-ridden condition for ten to twenty years.

Diagnosis.—In the typical cases there is no difficulty; the four cardinal symptoms—spastic paralysis of the legs, intention-tremor, nystagmus, and scanning speech—with or without weakness of vision, produce a clinical picture which is unmistakable. In its early stage there is, however, no other disease affecting the nervous system which leads to so many mistakes in diagnosis. The atypical cases may remain for years without showing the classical symptoms just mentioned; an excellent account of these is given by Risien Russell in Allbutt's 'System.'

Hysteria is with great difficulty eliminated in the early and in some atypical cases, even after the lapse of a considerable period. The points which settle the diagnosis are the pallor or atrophy of the disc, nystagmus, Babinski's sign, and early incontinence of urine; of these the presence of nystagmus is the most important, as this is never seen in its typical form in hysteria. The hysterical tremor is an intention-tremor, but, as pointed out by Gowers, it is much smaller in range, and the contraction of the antagonistic muscles is characteristic. Scanning speech, it must also be remembered, occurs sometimes in hysteria.

Locomotor ataxia may be mistaken for disseminated sclerosis, but the *permanent* loss of knee-jerk in ataxia and the absence of the extensive and characteristic volition-tremor are usually conclusive; moreover, syphilis plays no part in the causation of disseminated sclerosis.

Paralysis agitans has nothing in common but the tremor, and this is widely different from the jerky volition-tremor, as it is regular, rhythmical, limited in range, and occurs during rest, and the festinating gait of agitans is distinctive.

Cerebral tumour in the pons or crus produces sometimes incoördination, as seen in a jerky movement, evident only on voluntary effort, the tremor being identical with that of disseminated sclerosis; but the other signs of tumour, especially choked disc or optic neuritis and vomiting, will also be present. Cerebellar growths may produce nystagmus and incoördination; the drunken gait with other symptoms will aid in the diagnosis. When the sclerosis attacks the cerebellum the plaques as a rule are confined to the corpus dentatum.

Primary lateral sclerosis presenting typical spastic paraplegic symptoms, caused by myelitis of the cord or degeneration of the pyramidal tract, cannot be differentiated from the form of disseminated sclerosis which begins and remains for a long time confined to the spinal cord. In these latter cases there is no distinctive feature till an extension of the disease gives evidence of involvement of the cranial nerves, as optic-disc atrophy, nystagmus, and scanning speech.

Ataxic paraplegia may so closely resemble the spinal form of the disease under consideration, that differential diagnosis is impossible till the supervention of brain or cerebral-nerve symptoms, especially optic atrophy and nystagmus, prove the case to be one of disseminated cerebro-spinal sclerosis.

General paralysis of the insane is differentiated by the prominence of the exalted mental condition never seen in a marked form in sclerosis. The speech-affection is different also; it is slow and tremulous and hesitating, but never scanning, and the spasmodic twitching movements are confined chiefly to the muscles of the face, lips, and tongue, and are not strictly volition-tremors.

Infantile brain-injuries and *birth-palsies* affecting both motor hemispheres may closely simulate multiple sclerosis, but the history and progress of these are characteristic; and the same remark will apply to *Friedreich's ataxia*, which has always a marked family history.

Syringomyelia may be mistaken for multiple sclerosis, but the presence of trophic disturbances and the loss of pain- and heat-sensations, with the retention of tactile sensibility and the gradual development of a condition of progressive muscular atrophy, are distinctive, although nystagmus may be present in some cases.

Alcoholic and *mercurial* tremors are excluded readily by the history and by the presence of the constitutional symptoms produced by both these poisons and by the absence of nystagmus and speech-disturbances.

Choreic movements are not intention-tremors, but spontaneous irregular twitchings, which are, however, increased by attempts at voluntary movements and by mental excitement, and they exist independently of volition.

Prognosis.—This is always fatal, the only point of difficulty being in the forecast of its duration, as already mentioned. This is shorter in cases where the medulla is involved, as difficulty of swallowing may become serious at any time. The duration may be prolonged for years in cases which tend to stand still or improve for considerable periods. In most cases, however, after the full development of cerebro-spinal symptoms life is not likely to be prolonged beyond three or four years. Cystitis and secondary renal mischief are the common factors in the fatal issue, but death sometimes

follows the convulsive or apoplectiform seizures, which are attended by high pyrexia.

Treatment.—Beyond skilful nursing and scrupulous attention to the ordinary laws of hygiene, with most stringent antiseptic precautions where the catheter is necessary, and where bed-sores are present, little else can be accomplished.

Every known alterative drug has been tried, but there is no evidence that any marked benefit results. Arsenic and silver salts, iodides, and mercury are recommended; the writer thinks he has seen some temporary improvement follow the administration of the chloride of gold and sodium salt in doses of $\frac{1}{10}$ gr. in pill.

DISTOMIASIS.

(See under BILHARZIA, p. 105.)

DROPSY

This is the name given to the accumulation of serous fluid in the subcutaneous tissue or in the cavities lined by serous membrane.

When the accumulation into the subcutaneous cellular tissue is generally distributed over the body, the term *anasarca* is usually employed, and when restricted to the subcutaneous tissue of a localised region, as the legs or hands, or the loose tissue of the lung or glottis, the term *œdema* is often used; thus the terms 'general œdema' and 'anasarca' are synonymous, and they are often applied indiscriminately. When the accumulation takes place in the serous cavity of the abdomen, *ascites* is said to exist; *hydrothorax*, *hydropericardium*, *hydrocele*, *hydrocephalus*, and *hydrarthrosis* are the terms employed when the dropsy is confined to the pleura, pericardium, tunica vaginalis, cerebral ventricles, and joints. The fluid may be regarded as the liquor sanguinis or serum which has escaped from the blood-vessels. In the case of anasarca it is deprived of much of its albumin, but pleural and peritoneal dropsies are always rich in albumin, though the dropsical fluid of chronic hydrocephalus, consisting as it does of the normal cerebro-spinal fluid, contains only a trace of this substance. The fluid of dropsy differs from that resulting from inflammatory action in its lower specific gravity, in its markedly less tendency to coagulate spontaneously, and in its poverty of corpuscular elements and albumin.

Pathology.—Many points in the phenomenon of dropsy still require elucidation. The view of Cohnheim, that it results from an inflammatory change in the capillaries, is no longer tenable, since the investigations of Dickinson prove

that the resulting exudation or effusion found in the different regions of the body in dropsies does not possess the characteristics of an inflammatory product; and it appears equally certain that osmotic action plays but a very minor part in its production. In health the fluid which transudes through the capillary-wall is rapidly absorbed by the lymphatics and veins; the transudation remaining normal, it is obvious that any obstruction in the veins or lymphatics will so retard absorption as to lead to dropsy of the part. The lymphatics play an important *rôle* in aiding absorption when the venous obstruction is great; but when they are themselves blocked dropsy does not occur, the veins being able to carry off the exudation.

It is equally obvious that, the absorption-rate being normal, dropsy will result if there be a marked increase in the amount of fluid passing through the capillary-walls. In this case the accumulation takes place because the veins and lymphatics are unable to take up the increased or abnormal amount. The limitation of the lymphatic system is seen in animals in the experiment of Ranvier, which proved that no œdema occurred in the hind-legs of a dog after tying the ascending cava; but if the sciatic nerve was divided on one side the artery dilated and more exudation was poured out than the lymphatics could absorb, and œdema followed.

The results of experiments on healthy animals are, however, only to be accepted with great caution as applying to the elucidation of the problem of dropsy in the human subject under diseased conditions, because here two new factors are introduced—viz. an altered state of the blood and an altered condition of the lining membrane of the capillaries and small veins.

In the venous congestion produced in valvular disease by pulmonary obstruction, the pressure in the veins is greatly increased; this retards absorption, and dropsy is the result. Increased pressure is always the chief cause, but the other factors cannot be ignored, and here probably the explanation will be found of the anomaly that some cases of serious valvular disease may present no signs of œdema, whilst in other and apparently less severe cases œdema may be a prominent sign. The difference probably in these cases depends upon the presence or absence of changes in the endothelium of the small vessels.

Cirrhosis of the liver produces great obstruction to the flow of the blood in the portal vein and augmented pressure in all its radicals, the result of which is increased exudation of fluid into the cavity of the peritoneum, causing ascites. Thrombosis of the portal vein acts in the same way. Though the supervention of ascites is the general rule in both these affections, cases are occasionally met with in which ascites does not

occur, and after death the liver has been found in an advanced stage of cirrhosis, or the portal vein blocked by a thrombus. A similar state of affairs has been found in obstruction of the portal vein from malignant and gummatous tumours. Thus, in apparently the simplest cases of venous obstruction there are other factors at work above and beyond the presence of increased venous pressure leading to retarded absorption.

The dropsy of renal disease is not, as is usually accepted, the result of the accumulation of water in the system owing to the diminution of the amount passed through the kidney. Blocking of both ureters by calculi causes total suppression of urine, and no dropsy appears. The chief factor in renal anasarca is the alteration in the blood which causes it to transude more readily from the capillary-walls. Here it is a case not of diminished absorption, but of increased exudation; and the vascular condition is reversed, the pressure in the veins being low and the arterial tension high. The altered blood also increases peripheral resistance in the arterial twigs and capillaries, leading to cardiac hypertrophy, which may cause the disappearance of the dropsy after a time. At a later stage, failing compensation shifts the increase of pressure from the arteries and capillaries to the venous trunks, and dropsy appears. This late dropsy, especially in cases of cirrhotic kidney, is produced chiefly by the same mechanical factors which cause dropsy in mitral regurgitation; but probably all the factors which cause dropsy are at work in such a case—viz. (1) venous pressure causing retarded absorption; (2) altered blood inducing increased exudation; (3) a change in the endothelium of the small vessels leading to a freer transudation; (4) possibly a higher arterial pressure in the capillaries which would also tend to increase the outpour; and (5) possibly the intervention of more or less vasomotor paralysis.

The œdema of anæmia and chlorosis obviously depends upon a watery state of the blood and probably secondary changes in the vascular walls; and a similar explanation meets the anasarca of purpura, gout, and diabetes. Beri-beri dropsy is the result of blood-changes plus vaso-motor and endothelial alteration. That local œdema in urticaria and allied states depends upon a rapid alteration in the blood is demonstrated by the ease and rapidity with which it can be removed on increasing the coagulability of the blood by the administration of chloride of calcium.

The etiology, diagnosis, prognosis, and treatment of the different varieties of dropsy will be found under the headings of the primary diseases which produce them.

Angio-neurotic œdema is the name given to a rare form of transient local anasarca which suddenly appears in circumscribed areas of the body, often on the face, hands, throat,

feet, or the genitals. The swelling is usually accompanied by considerable local discomfort, and generally by very severe intestinal pain or colic, followed often by vomiting. The attacks may come on with great regularity every few weeks, or oftener; and though usually they pass off without leaving any trouble behind them, occasionally death has been known to occur from the œdematous swelling of the larynx. The most interesting feature in this rare condition is its well-marked hereditary character, which may be traced through many generations.

Its pathology is probably that of a vaso-motor neurosis; but the relationship of the œdematous swellings to those seen in severe urticaria, and the combination of gastro-intestinal disturbances, are suggestive of some toxic condition. The affection is almost beyond the reach of treatment, though Osler states that he has obtained satisfactory results from the prolonged administration of nitroglycerin.

DYSENTERY

Under this name are included several forms of infective inflammation of the colon characterised by the clinical symptoms of frequent bloody, mucous, or serous motions, accompanied by severe griping pain with tenesmus, and presenting after death anatomical changes of a catarrhal, ulcerative, diphtheritic, or gangrenous nature.

Etiology.—The disease occurs in true *epidemic* form, breaking out in many places at once, generally in the autumn season in temperate climates, independent of any spreading from endemic cases. *Endemic* dysentery is widely distributed from the temperate regions to the equator, flourishing in marshy, badly drained districts in its home in the tropics. The disease is becoming much less frequent in the presence of improved sanitary conditions, especially in temperate regions. From endemic foci it may spread to the extent of a minor epidemic under suitable conditions, as *overcrowding*, *famine*, and *war*. Thus in the British Isles it was formerly not uncommon in military barracks, hospitals, and asylums, and it still makes its appearance amongst troops in the field, as evidenced by the late Boer war and Russo-Japanese campaign.

Its prevalence in temperate climates is intimately associated with contaminated water-supply, and many instances are on record where the importation of a case has been followed by an outbreak spreading over a wide area, as is often the case in cholera and typhoid fever. In these instances it is probable that the pollution of the soil by the excreta and the ultimate contamination of the drinking-water are the main factors in

its spread. Causes, such as serious chilling of the cutaneous surface, which lower the resisting power of the intestinal membrane, markedly aid in the spread of the disease, and the study of many epidemics leads to the conclusion that organisms other than the so-called specific virus play an important part in the spread of the disease. Thus decomposing food, impure water, over-ripe fruits, and all agents which tend to produce catarrhal diarrhœa, appear to pave the way by increasing the virulence of the bacillus coli, streptococci, and staphylococci normally present in the bowel; and it cannot be doubted that these organisms play an important *rôle* in the suppuration and necrosis of the colon when once started by the specific virus.

It appears also almost certain that the dysentery arising in military camps, and that which occurs in districts supplied by drinking-water contaminated by fæcal matter, do not necessarily depend upon the introduction into the body of the discharges from patients suffering from dysentery. The period of incubation varies, and in the acute non-amœbic form it may not exceed forty-eight hours.

Morbid Anatomy and Pathology.—There is a general resemblance in the lesions observable in the colon in all the varieties of dysentery, and much confusion is caused by describing as distinct types of the disease what are probably only different stages or varying degrees of severity of the same inflammatory process.

The two affections—*amœbic* and *non-amœbic* dysentery—may possibly be found to be after all but modifications of the same diseased action.

Bacteriology.—Amœbic dysentery.—The specific amœba of dysentery (*Entamœba dysenterica*) has been recently differentiated beyond question from the harmless amœba (*Entamœba coli*) often found in perfectly healthy individuals in certain localities by Schaudinn, whose researches have been confirmed by Craig. These observers have frequently found both of these distinct species in the bowel of patients suffering from dysentery. The *E. coli* plays no *rôle* whatever in dysentery, and it has been demonstrated to be non-pathogenic; it is much smaller; it occurs in encysted forms, is of an opaque grey colour; its protoplasm does not show when at rest distinct separation into ectosarc and endosarc; it is never seen to contain more than one or two red blood-discs; it seldom shows any vacuoles, and has a very limited degree of motility.

The specific *E. dysenterica* (also known as *E. histolytica*), when obtained from fresh dysenteric stools, measures in diameter from 25 to 35 μ; it is actively mobile, throwing out long pseudopodia upon the warm stage, and usually shows a distinct tinge of green. One of its most characteristic features is the highly refractile nature of the scanty ectosarc in the

pseudopodia, which also stains intensely. It exhibits numerous vacuoles and is often filled by red blood-discs, and it possesses a large lateral nucleus, as may be seen by examining a pellet of fresh mucus immediately after its expulsion from the bowel. The parasite has also been found in the pus from hepatic abscess occurring in tropical dysentery, and in water contaminated by dysenteric stools.

In *non-amœbic* dysentery various organisms, especially streptococci, staphylococci, and bacillus coli, are always to be seen, but one short bacillus has been found always present by Shiga, who insists upon its specific character. It resembles the bacillus of typhoid, and its cultures agglutinate with the serum obtained from dysenteric patients. It is known as the *Shiga-Kruse bacillus*, and it appears to be identical with the organism described by other investigators under the name of the *bacterium coli dysenteriæ*. There are some grounds for believing that this is a modified or virulent form of the bacillus coli, as formerly the amœba dysenterica was supposed to be a virulent form of the ordinary amœba coli. This hypothesis, if accepted, readily explains the fact previously mentioned, that the dysentery of war and famine, and that produced by drinking-water polluted by fæces, may occur without any contamination from the evacuations of patients actually suffering from dysenteric symptoms. Upon the other hand, Hiss has pointed out that this bacillus differs from all the allied strains by not fermenting mannite. Of the *non-amœbic* form of the disease, three varieties are described founded upon the characters of the bowel-lesion. In *catarrhal* dysentery the mucous membrane is deeply injected, the solitary follicles are prominent, spots of hæmorrhage are seen in the swollen mucosa, which is covered over with a layer of bloody mucus. When the inflammatory process has been established for some days, *necrosis* or *ulceration* begins to show itself in the mucosa, commencing in small spots which spread into broad, transversely placed, deep ulcers. Sloughs and gangrenous patches of a dark grey colour are often present between the ulcerated areas or in different portions of the bowel. Though the usual site is either at the cæcum and adjoining portion of the ascending colon, or at the sigmoid, and the upper part of the rectum, the catarrhal, ulcerative, or gangrenous process may attack the entire extent of the large bowel, and it usually shows different stages and degrees of intensity in different portions of the gut. As a rule, the lower parts of the colon (rectum and sigmoid) are most deeply involved. In intense examples of the disease the entire colon may be converted into a flaccid, gangrenous, foul-smelling tube, with its calibre little interfered with, though on section the sloughing process may be found to extend through all its coats down to the peritoneum.

In the so-called *diphtheritic* dysentery, as the catarrhal inflammation proceeds the mucous and sub-mucous coats become the seat of an exudation which converts the colon into a rigid tube of narrow calibre. The sloughing process supervenes, destroying both coats, and often involving the muscular layer as well.

Where the diphtheritic exudate only involves the surface of the mucosa and does not penetrate the sub-mucous layer, gangrenous action does not follow, but the process ends in ulceration, and is described as *croupous* dysentery, though the ulcers may afterwards eat their way down to the peritoneum. The morbid anatomy of *amœbic* dysentery does not markedly differ from the catarrhal form. The cæcum and ascending colon are chiefly affected, though the entire extent of the colon may be involved, and the disease may extend into the small intestine. There is much thickening always present, mainly in the form of a general œdema of the mucosa, with nodular areas of circumscribed induration. Over some of these, necrotic spots appear, which soften and lead down to ragged ulcers in the sub-mucous coat, roofed by overhanging mucous membrane and communicating by sinuses with other sub-mucous ulcers. From these the amœba in active movement may be obtained when the examination is made soon after death. The ulcers may penetrate and undermine the muscular layer, large portions of which, with attached mucous membrane, may be passed during life in the evacuations, but the primary seat of the lesion is in the sub-mucous coat, though the peritoneum is often thickened, and adhesions are formed between neighbouring coils of bowel.

The same diphtheritic or croupous exudation as has been mentioned as occurring in the non-amœbic form of the disease is sometimes observed, and occasionally large gangrenous areas exist, involving a considerable extent of the bowel.

Abscess of the liver is commonly met with, and perforative peritonitis is not rare. The spleen and pancreas are enlarged and softened, perityphlitis, and pylephlebitis are often met with, and in both the amœbic and bacillary types the abdominal lymphatic glands are enlarged and softened.

Chronic dysentery presents the *post-mortem* changes which are the result of the inflammatory process seen in the acute cases. Ulceration may have progressed to such an extent that repair causes bands, plaques, or extensive constrictions, or even obstruction of the bowel, especially in the region of the sigmoid. Open ulcers may be found alongside the cicatrices of those which have healed, and indurated masses of tissue may lie between. Generally, unhealed ulcers are present above the constrictions. Atrophy of considerable portions of the wall of the bowel is met with in the intervals between thickened and puckered indurated areas,

and matting of the intestines is common, with enlargement and pigmentation of the mesenteric glands. Sometimes the exudation poured out between the layers of the bowel-wall becomes so dense that a portion of the bowel when removed may appear patent like a piece of hose-pipe.

Further research may justify the division of dysentery into the three classes made by Ford: (1) bacillary; (2) amœbic; (3) mixed. With our present knowledge, it is safer to regard them all as mixed, and to divide them into amœbic and non-amœbic.

Symptoms.— *Non-amœbic dysentery.*—The onset varies greatly. In times of epidemic the invasion is often abrupt and severe. The general rule in endemic cases is for an apparently innocent diarrhœa of two or three days' duration to be followed by some griping pain and peculiar motions. The first evacuations are loose and semi-feculent, but they soon change and present an appearance which is amongst the most characteristic of the clinical features of the disease; all traces of fæces disappear from them, the motions being composed of mucus, blood, and serous fluid. Colicky pain of a severe kind precedes each movement of the bowel, accompanied by prolonged and distressing tenesmus or straining, which is greatest when the sigmoid or upper part of the rectum is the site of the affection; and dysuria is often present. The desire to relieve the bowel becomes frequent and sometimes continuous, so that the patient spends the greater portion of the time on the night-chair. As many as a hundred motions, or even double this number, may be passed during twenty-four hours; but in mild cases about a score of small jelly-like blood-stained evacuations of a few ounces each are passed. As they become more frequent they lose their slimy character, decrease in bulk, and become more fluid, consisting of a reddish serous and highly albuminous liquid, with an offensive but non-feculent odour. At this stage they resemble a spoonful of the juice or scrapings of meat, and consist of blood-cells, pus, and minute sloughs or shreds of mucous membrane and broken-down tissue, mixed with food. With the continual tenesmus the anus becomes excoriated and tender, and prolapse often occurs, accompanied by strangury, and much blood may appear in the motions. In the early stage the constitutional symptoms are slight, though there may be some restlessness and malaise and furred tongue, with increase in the pulse-rate. Fever is usually absent, and rarely is there more than a moderate elevation of the temperature all through the attack, unless malaria or other complication supervene. As the motions become more fluid, offensive, and numerous, the constitutional symptoms deepen: the tongue becomes dry, thirst increases, the pulse gets rapid and weak, vomiting may set in, and

emaciation is evident, with complete loss of appetite. There is marked tenderness on pressure over the course of the colon. In mild cases the symptoms may not pass beyond the catarrhal stage, showing the jelly-like motions, when in about ten days the first signs of improvement set in by the gradual appearance of distinctly feculent matter in the evacuations, which become less frequent and firmer in consistence.

In the severer cases the fluid offensive motions may appear as early as the third or fourth day, and, especially in cases occurring in tropical or sub-tropical countries, the disease may progress so rapidly that death may take place within a hundred hours of the onset.

When improvement sets in after the motions have become liquid and offensive, the process is generally very gradual, the first change noticed being the appearance of feculent material in the evacuations. When the attack passes through the two stages just described without showing signs of improvement, the third or *gangrenous* stage may be expected. This may rapidly supervene upon the mildest catarrhal type, or it may set in with acute symptoms of great severity, as in the diphtheritic variety of the disease, where the catarrhal stage may be very short.

The advent of the gangrenous stage is marked by an obvious change in the motions; these become greenish or brownish-black, contain pus and dark-coloured sloughs of various dimensions and characters, according to their origin and extent in the mucosa or sub-mucosa. The most obvious feature of the motion, however, is its horrible gangrenous odour. Sometimes large quantities of blood are passed, and tubular sloughs from a few inches to a foot in length may be found in the evacuations.

With these changes in the appearance of the motions the constitutional symptoms become grave: rapid loss of strength, emaciation, and great prostration follow.

The temperature, which may have reached an elevation beyond the normal of 3° or 4° F. in cases with an acute onset, now falls, and in the majority of cases at this stage a sub-normal temperature is the rule. The pulse gets quick and thready, the tongue dry, glazed, and dark, and the features pinched; vomiting and hiccough may appear, the anus relaxes, tormina and tenesmus disappear, the urine becomes scanty or suppressed, the extremities become icy cold, and the patient sinks from exhaustion.

All cases which reach the gangrenous stage do not succumb. Even after the passage of large sloughs, improvement may set in which ends in slow recovery, or the disease passes into the following stage.

Chronic dysentery may begin as a recurring diarrhœa, alternating with attacks of constipation, or it may be the direct

sequel to an acute attack, which is the commoner form. It is much more frequent in the tropics than in temperate zones, and it is often of amœbic origin ; but the most protracted cases are often of the non-amœbic variety. The symptoms are those of the ordinary catarrhal type, with some colicky pain and tenesmus, accompanied by blood-stained mucoid motions. These cease for periods of several days or weeks, during which healthy feculent evacuations may be passed. Sub-acute exacerbations follow these quiescent periods, and these may exhibit all the changes seen during the course of the acute affection, even to the extent of the passage of dark-coloured, gangrenous, offensive motions containing sloughs, pus, and blood, accompanied by much tormina and tenesmus.

Gradually appetite and digestion fail, emaciation to a remarkable extent follows, with anæmia and loss of strength, and the patient may slowly sink from exhaustion or succumb from abdominal obstruction caused by cicatricial narrowing of the bowel, or from a perforative peritonitis, or from amyloid disease of the viscera. The disease may last for many years. In one case under the writer's observation, contracted in the tropics, the patient dragged out a miserable existence for twenty years.

Amœbic dysentery is sometimes wrongly called tropical dysentery, as it is not confined to the tropics, but is often met with in temperate regions ; moreover, the bacillary type of the catarrhal or diphtheritic disease is also met with in the tropics, though more common in colder regions. As already mentioned under the head of Morbid Anatomy, the pathological process in dysentery consists of a mixed infection. The streptococci found in endemic and epidemic dysenteries of temperate zones, in both the catarrhal and diphtheritic types, are always present, working in conjunction with the specific amœba in the amœbic dysentery of hot climates. This is the probable explanation of the resemblance of the symptoms, or of the supervention of gangrenous or diphtheritic characters upon the amœbic variety of the disease ; and many authorities maintain that the symptoms of both types often so closely resemble each other that clinical differentiation is impossible.

This form of dysentery varies widely in its symptoms and onset, and for a considerable period in its course it presents clinical features which differ upon the whole from those commonly met with in the non-amœbic form. It is, as a rule, slower in its onset, more irregular in its course, much more protracted in its duration, and the complication of liver-abscess is much more frequently found in association with it.

In the grave cases the onset may be as sudden as in the severe forms of bacillary dysentery ; but as a rule it is gradual, commencing with a painless diarrhœa, alternating with constipation, and without fever, and with little or no tenesmus.

The motions for a considerable time may contain liquid yellow fæces; mucus is always present, but blood is not, as a rule, an obvious feature. The periods of quiescence and diarrhœa are very variable in their duration, and all observers agree that in many instances the symptoms may be so mild and treacherous that the presence of the disease is not suspected till attention has been directed to the hepatic suppuration.

Anæmia, emaciation with an icteroid tinge, loss of appetite, and vomiting occur during the period of diarrhœa; as this subsides, the patient regains somewhat his normal condition for a time. The duration of the disease may extend over many months, and each attack of diarrhœa tends to become more severe than the former. Some pain and slight tenesmus appear, and the stools show small shreddy, necrotic, gelatinous masses, shed from the intestinal ulcers, which always contain amœbæ. At any period in its course, and from the commencement of the acute grave cases of sudden onset, gangrenous characters may be observed in the motions, which may contain sloughs and give out a markedly putrid stench. The clinical picture at this stage closely resembles that seen in the gangrenous form of non-amœbic dysentery. Recovery is always tedious; it may set in at any stage, but the essential character of the affection is towards chronicity; the tedious cases run into the groove already described under 'Chronic Dysentery.' Life may be cut short by perforative peritonitis or hepatic abscess or secondary pulmonary suppuration. The first signs of improvement are noticeable in the diminution of the frequency of the stools, which may number twenty to thirty daily in severe cases; with their diminished frequency the odour, consistency, and other abnormal characters tend towards the normal. But it must be remembered that such improvement may but mark one of the quiescent periods which is to be followed by a more acute exacerbation later on.

Sequelæ and Complications.—*Abscess of the liver*, as already mentioned, is more commonly the result of amœbic dysentery, which furnishes most of the cases of the large single abscess known as the *tropical* abscess. When the abscess follows the bacillary or non-amœbic form, it is usually multiple or of the pyæmic type. (See under 'Liver Abscess.')

Malaria is not an uncommon complication of dysentery in the tropics, and malarious dysentery is the name sometimes given to a type in which the symptoms of both diseases are inseparably blended. Peritonitis and perforation of the bowel are frequent in chronic amœbic dysentery. Arthritic or rheumatic joint-troubles, and paralysis or neuritis, occasionally appear as sequels or complications.

Diagnosis.—The frequency and the specific characters of the serous stools showing the total absence of feculent matter,

the tormina and tenesmus, form a clinical picture easily distinguishable from *ulcerative* and *mucous colitis*.

Enteric fever is simulated by severe cases of the non-amœbic form which are about to show the diphtheritic or gangrenous type ; but a few days' delay in such cases will establish the characteristic change in the motions, with their sloughs and their horrible gangrenous stench. The agglutination of the Shiga-Kruse bacillus with the serum of the patient is a valuable diagnostic point. Amœbic dysentery is recognised with certainty by the detection of the protozoon parasite always easily found in the stools, and by its chronic irregular course.

Cancer and *syphilitic ulceration of the rectum* may be mistaken for chronic dysentery, but a digital examination and the history suffice to distinguish them.

Prognosis.—This is very variable, and differs much in different epidemics. Speaking generally, the mortality of late years has been steadily declining in the face of improved hygienic measures.

In catarrhal cases, with jelly-like stools, the prognosis is good ; but it deepens with the change to the serous, especially in the presence of early treatment. In the gangrenous stage the prognosis is always grave, even in the absence of sloughs ; but recovery may occur in all stages of the disease if the resisting power of the patient is good ; the supervention of typhoid symptoms, and especially of hiccough, renders recovery highly improbable. It must never be forgotten that gangrenous action may supervene at any time in the most innocent-looking cases ; and this is especially to be feared in the amœbic cases, where also the hepatic and pulmonary suppurations and the special tendency to peritoneal complications render the prognosis one of great gravity when signs of these events show themselves.

The prognosis in chronic dysentery will depend largely upon the presence of changes in the bowel tending to produce obstruction, and also on the amount of cachexia present and upon the duration of the disease.

Treatment.—The *prophylaxis* of dysentery, especially in war and amongst travellers in the tropics, is a question of vital importance. In addition to general hygienic precautions, in the presence of endemic or epidemic dysentery the dejecta should be destroyed by fire or disinfectants and buried deeply in the soil ; bed-linen should be kept scrupulously clean, and all drinking-water boiled before use. Muscular exhaustion, chills, irregular living, and the use of alcohol should be avoided, and attention should be given to the condition of the bowel, especially if diarrhœa or a marked tendency to constipation exist.

As soon as dysenteric symptoms show themselves, the patient must take to his bed and be fed by small amounts

of liquid food frequently administered. Milk, fresh or peptonised, is the best; but where milk is not well borne, chicken-jelly or meat-juice may be given instead.

Serumtherapy has produced good results in the hands of Rosenthal, who has immunised horses by the injection of cultures of the Shiga-Kruse bacillus. The serum of such immunised animals, he claims, reduces the mortality by a half and shortens the attack, and if given at the onset ensures always speedy recovery. Shiga claims even better results; and Todd's serum, prepared by immunising horses with injections of the bacillus cultivated upon alkaline broth, has a powerful antitoxic action, less than $\frac{1}{50}$ min. protecting a rabbit against twenty lethal doses of the toxin. Wright's vaccine treatment has been also successful.

The clear indication in any form of dysentery is to administer an intestinal antiseptic which will destroy the parasite, but it cannot be said that such has as yet been discovered: salol, naphthalin, naphthol, benzo-naphthol, and others have been tried with very varying success. Calomel as a bowel-disinfectant has given better results than any of these substances; but, though apparently very efficacious in some epidemics, it is valueless in others, and the general practice is only to employ it as an adjunct to other treatments when these have failed alone. One grain may be given hourly for six or eight times in combination with the drug to be next mentioned, when it has failed alone.

Ipecacuanha, though purely an empiric remedy, still affords the best results obtainable in bacillary dysentery, though of little use in the amœbic form unless when combined with other drugs. As soon as the diagnosis is clear, a dose of 30 grs. should be administered in cachet form after a couple of hours' fast, and the patient should get no food for two hours after, but be kept perfectly still in the recumbent posture with the view of avoiding vomiting; 20–30 mins. of laudanum just before the administration of the ipecac. often assists in its retention. Smaller doses are useless, and the above amount may be given three times a day, or 15 grs. may be given six times daily. A large sinapism applied over the abdomen ten minutes before the administration of the first large dose may be tried to prevent vomiting and nausea. De-emetinised ipecac. has not gained in favour. The ipecac. treatment should be kept up for two or three days, when the dose may be lessened or the intervals lengthened. If no improvement follows, small doses of calomel may be combined with it.

Salines, as sulphate of soda, given in 6-dr. doses morning and evening, have often been proved beneficial, and castor oil, on the same principle of flushing out the intestine, has often been found of great value, especially in the treatment of children's dysentery, when given after minute doses of

calomel or grey powder. The saline treatment may be used in conjunction with ipecacuanha.

There is much difference of opinion about *irrigation* in acute dysentery. In some cases it relieves the tenesmus. A quart of normal saline solution should be slowly injected into the rectum with the patient's head lowered and his hips raised ; a soft long tube should be used. This amount, after ulceration and gangrene had set in, might cause serious damage ; therefore, if irrigation is to be employed in acute dysentery, it should be in the early stages of the disease.

Symptoms as they arise must be combated by appropriate agents. Abdominal pain will call for poultices, fomentations, and opium ; tenesmus and dysuria for small laudanum enemata or larger injections of pure olive oil ; hæmorrhage from the bowel can be controlled by adrenalin.

Amœbic dysentery is best treated with salines by the mouth, in conjunction with full doses of quinine ; ipecac., combined with calomel, may be tried when these fail. Irrigation is more clearly indicated in this form of the disease, and the best solution is one of quinine ; 1 drm. of the acid hydrochloride dissolved in 3 pints of water may be injected, or the same proportions of eucalyptus gum may be used twice daily. Corrosive sublimate or copper sulphate 2 grs. to 20 oz., or nitrate of silver 20 grs. to 20 oz. may be employed.

Chronic dysentery must be treated upon the same general principles. Change of climate is essential in most cases, and during the diarrhœal exacerbations the patient should be put to bed and treated with a milk and strong soup diet. Concentrated easily digested food must be given at short intervals, the main element in the treatment being the feeding. Rarely will ipecac. be indicated, but intestinal antiseptics like salol may be freely given.

Irrigation with solution of nitrate of silver or sulphate of copper 10 grs. to 40 oz. may be freely used, and ulcers within reach may be touched with the solid stick or crystal. Diarrhœa will call for astringents like tannalbin, and constipation will be met by mild laxatives like sulphur, olive or castor oil.

Obstruction of the bowel is best treated by emollient enemata, but surgical procedures may be demanded.

The treatment of abscess of the liver will be found detailed under ' Liver Abscess.'

DYSPEPSIA, SIMPLE

This is but a synonym of gastric indigestion as evidenced by pain, distension, vomiting, anorexia, acid eructations, or other symptoms indicative of the arrest of the digestive function or of fermentation, or of some perverted action in the normal digestive process. It is a symptom of all the

organic diseases of the stomach, as ulcer, cancer, atony, atrophy, pyloric obstruction, the presence of adhesions, and the various forms of inflammation of this organ.

When these are excluded, there remains a large group of conditions which in our present state of knowledge we must define as functional. Much confusion has resulted from the indiscriminate lumping of the different members of this group under the rather arbitrary nomenclature of 'Gastric Neuroses,' to the complete exclusion of the term 'Dyspepsia.' It cannot be denied that many of the cases of dyspepsia or indigestion met with in practice are obviously caused by errors in diet or irregularities of feeding, and the confusion is not lessened by grouping such cases under the heading of 'Gastritis,' since the gastritis is manifestly the result and not the cause of the indigestion.

There are still good grounds for reserving the term 'dyspepsia' for a type of case often to be met with, in which there is no organic or inflammatory lesion, and in which the nervous apparatus presiding over the digestive process is not in an abnormal condition.

ACUTE DYSPEPSIA

Etiology.—(1) Some irritating substance is introduced into the healthy stomach, generally in the form of improperly cooked food, as baked fat meat or pork, food which has commenced to undergo putrefactive change, warm freshly baked bread or cakes, improperly made pastry, unripe fruit, acid beer or wine, or a large quantity of iced water, and an attack of indigestion follows. (2) The food may be normal in every respect, but taken in such an amount as the healthy stomach is unable to digest. (3) The quantity and quality being normal, it may be hastily bolted in lumps without mastication, so that the gastric juice only comes into contact with the outer surface of each mouthful, and the same result follows. (4) The intervals between the meals may be so short as to prevent the stomach being emptied before another supply of food is introduced, or the introduction of a normal meal may bring on an attack of acute dyspepsia after a prolonged and exhausting fast.

Symptoms.—If the irritant be capable of producing a severe local impression the symptoms of pain and discomfort come on rapidly and eventuate in speedy emesis, as in irritant-poisoning. More usually, however, a period of one or two or more hours intervenes after eating before the first feeling of discomfort is noticed. This is generally a sensation of distension, which may pass into acute pain in the region of the stomach; it is obviously caused by an accumulation of flatus, which may be expelled with force through the mouth; with it often are acid eructations, small quantities of a burning liquid being regurgitated into the mouth or back of the throat at

intervals. This fluid is of such an irritating sour nature as to set the teeth on edge, and in cases where fat baked meats have been eaten it consists mainly of butyric acid in such concentration that it may destroy the pharyngeal epithelium. After a late supper of such over-cooked rich meat, the patient may awake during the night seriously ill with acute gastric pain, followed soon by severe vomiting; the writer has seen alarming spasm of the glottis caused by the result of the corrosive action of the acid contents of the stomach, as they passed over the epiglottis. With the occurrence of vomiting there is speedy relief to all discomfort, flatulent distension, and pain, though sometimes nausea may remain with headache and malaise. Not infrequently smart purging follows.

The vomit consists of the food very slightly altered. It has a disgusting sour odour, though as a rule it contains little or no hydrochloric acid, the acidity being caused by the presence of the organic acids, the product of the butyric and lactic acid fermentation. There is no time for furring of the tongue unless the attack has been prolonged, and, as a rule, the appetite speedily returns.

Treatment.—The occurrence of vomiting often renders interference unnecessary, but in severe pain and distress it may be imperative to induce vomiting by tickling the fauces or administering copious draughts of lukewarm water or ipecacuanha. Where there is evidence of great acidity in the stomach, a full dose (a teaspoonful) of bicarbonate of soda in a tumblerful of water gives immediate relief. The subsequent treatment consists in total absence of all food for at least twelve hours. Aërated water may be taken frequently; and where purging has not occurred a dose of calomel or a smart saline may be beneficial.

CHRONIC DYSPEPSIA

As already stated, there is room for the recognition of this affection after the elimination of all cases depending upon organic or inflammatory conditions of the stomach, and of the series of cases classified as gastric neuroses.

Etiology. — All the causes just mentioned under 'Acute Dyspepsia' may be at work in keeping up a chronic degree of discomfort and pain, the result of imperfectly performed digestion. Thus, errors in the quantity, quality, cooking, and mastication of the food, as well as irregularity in the hours of eating, are to be constantly met with amongst persons presenting themselves for relief of stomach-troubles, and when these errors are corrected the symptoms rapidly disappear. Where such factors have been long in operation, and the violation of health-laws has been very protracted, there are good grounds for believing that a chronic inflammatory process of a parenchymatous and interstitial nature, involving

the entire gastric mucous membrane, may supervene, which may persist long after the removal of the exciting cause; such cases may be properly classed under 'Chronic Gastritis.' It is often comparatively easy in practice to differentiate these two conditions by a study of their history and an investigation of the habits of the patient. Sometimes, though rarely a continuance of the vicious methods of dieting and irregularities in the intervals between meal-hours may set up or induce local neurotic disturbances, which may obstinately linger after the patient has been taught to diet himself rationally. Such cases may fairly be considered as falling within the category of the gastric neuroses; this is exemplified in the dyspepsia caused by excessive tea-drinking.

In addition to the causes mentioned under 'Acute Dyspepsia,' there is a very common etiological factor at work in the production of chronic indigestion, the importance of which can hardly be exaggerated—*i.e.* the presence of microbes in the hollow cavities of the molar teeth. These are produced in myriads, and are mixed with the food during mastication, and on their introduction into the stomach play an important part in the arrest or perversion of the digestive function, as is demonstrated by the speedy disappearance of all dyspeptic troubles after the mouth has been cleared out by the dentist. Filthy dental plates act in a similar manner, though to a less extent. Owing to individual peculiarities, a chronic disturbance of the digestive process may be set up in one patient by articles of food which another may partake of with impunity.

Causes of chronic indigestion, such as excessive fatigue immediately before or after eating, excessive mental activity, shock, or emotion, strictly speaking, belong to those cases grouped under the gastric neuroses.

Symptoms.—These are of a similar character, though they are less in intensity than those met with in the acute condition. As a rule, a considerable time elapses between the meal and the discomfort which follows; in mild cases this may not exceed a feeling of distension or oppression in the epigastrium, the food causing a sensation of weight, owing to the total arrest of the digestive process.

Pain is a common symptom; it is of a dull, diffused character, referred to the epigastrium, and sometimes, especially when acidity is the cause, it is described as of a burning nature; it lasts for a considerable time, and gradually passes away as the contents of the stomach flow through the pylorus, after which it may travel towards the lower regions of the abdomen. It is sometimes described as *gastralgia, cardialgia, gastrodynia*, or *heartburn*. The term 'gastralgia' should be restricted to the paroxysmal pain of a neuralgic character, which is generally independent of food, and is characteristic of a gastric neurosis. The term 'heartburn' should be reserved for

the gastric pain, or burning sensation, which originates at the junction of the stomach and œsophagus, and which is felt at the lower end of the sternum; it is a common symptom in most varieties of dyspepsia, and is often associated with *pyrosis*, or water-brash. This consists of the regurgitation of an alkaline fluid, consisting mainly of saliva, which has accumulated at the lower end of the gullet; the term is also applied to the acid eructations or regurgitations which come from the stomach.

Flatulence and *acidity* are common features, especially the former. The latter may be entirely absent; when present it is usually owing to an excess of the organic acids produced by fermentation, and not, as in some gastric neuroses, caused by hyperchlorhydria. The flatus may be expelled forcibly through the mouth, or it may reveal its presence by borborygmi, or splashing sounds in the stomach or intestines, or it may be passed by the anus. The source of the flatus is doubtful; in many cases, however, it is clearly the result of fermentation, but often it is the result of air swallowed in the *voluntary* action of rifting, or belching up some of the gas which is normally present. This habit is a common cause of dyspepsia, or rather a constant cause of the most trivial cases of indigestion being converted into the most obstinate dyspepsia. It is difficult to convince the dyspeptic that every time he makes this voluntary effort he swallows more air than he expels, and so distends his stomach as greatly to interfere with its function. If a person in health tries to expel the gas from the stomach in this manner for a few times, he will soon find that there is a difficulty in stopping it, and that the stomach, and apparently the entire abdomen, may become distended. It is surprising how chronic dyspepsia in some patients may be made to rapidly disappear once they have learned the evil of this habit. In such cases it is an obvious mistake to regard the excessive flatulence as evidence of a neurosis. In swallowing saliva, a considerable volume of air accompanies it into the stomach, but this as a rule causes no inconvenience.

Appetite is variable; as a general rule it is not much affected, but sometimes it may be markedly increased.

Nausea is a common feature in long-standing cases which are passing into the phase of chronic gastritis, and usually there is then loss of appetite.

Vomiting is sometimes present; it may come on at any stage, but more commonly two or three hours after a meal. The vomit consists of the food, often but slightly changed; it is generally acid from butyric, lactic, and acetic acids, but free hydrochloric is generally much diminished. It may be delayed for six or more hours, at which period the constituents of the meal should have passed into the intestines. In the

chronic dyspepsia of alcoholism, morning vomiting, nausea, or *retching* (*i.e.* ineffectual attempts at vomiting) are characteristic symptoms.

The *tongue* is usually furred. The old-fashioned division of chronic dyspepsia into *atonic* and *irritative* was based upon the character of the tongue, which in the atonic variety was large, indented at the margins by the teeth, and covered over with a moist white fur; while in irritative dyspepsia the organ was small, red, clean, and often dry. The state of the tongue was, and is still, supposed to be an index of the condition of the gastric mucous membrane. Foulness of the breath, a bad taste in the mouth, and obstinate constipation are usually present.

Nervous symptoms are sometimes observable, from irritability of temper and restlessness, to languor and inability to fasten the mind upon any subject; but rarely is very marked depression of spirits and melancholy noticeable. These symptoms, if present, commonly pass away immediately after eating, to return in their most acute form with the arrest or perversion of the digestive function.

Vertigo and headache are not uncommon; the urine is scanty, and generally deposits urates or phosphates; functional disturbance of the heart may be a prominent feature where there is much gastric distension from flatus. Often the body-weight is little interfered with.

The condition cannot be said to present any special morbid anatomy, unless it has been of such severity and long standing as to induce inflammatory changes in the gastric coats, when the atrophic or sclerotic features of chronic gastritis may be superimposed—in which case the affection must be regarded not as simple dyspepsia, but as chronic catarrh of the stomach.

Its duration is indefinite as long as the exciting causes continue, though long periods of relief are common, in which most or all of the symptoms may disappear, to return again upon indiscretions in eating and drinking, or upon irregularities in the times of meals.

Treatment.—The indications for the prevention and treatment of the simple chronic dyspepsia just described are obvious. The cause is to be found out in each case, and the quantity, quality, and method of cooking of the food are to be investigated and the errors therein corrected. The intervals between the meals are to be regulated, and periods of repose after eating prescribed, in which neither mental nor physical exertion is to be indulged in. The character of the liquids consumed at meals, as well as their amount, should be investigated, and rules laid down for the patient's guidance in this detail are of importance. The state of the teeth should be carefully attended to, and not only disinfection of the mouth insured, but assistance must be rendered when necessary by

artificial teeth where mastication is at fault. The patient should be warned of the necessity of slow and complete comminution of all food in the mouth before swallowing. In the author's 'Dictionary of Treatment' (4th ed.), pp. 237–247, the details of the management of dyspepsia are discussed at length, and need not be here reprinted. One of the most serious mistakes which the young physician is liable to make is to fall into the groove of prescribing a 'cut-and-dry' restricted dietary for every case of dyspepsia. The great majority of cases coming under notice for the first time are found to be living upon a diet-roll from which one article after another has been excluded because of some mistaken notion about its indigestibility, till the list has become so restricted that healthy life cannot be maintained unless generous additions be insisted upon. One rule to which there should be no exception must be insisted upon—*i.e.* that *re-cooked* meat is never to be taken; and *baked* meat must be forbidden.

Every physician can testify to the truth of Flint's statement that 'in a great number of cases in which persons have been sufferers for years on a regulated diet, health has been speedily regained by simply eating in accordance with appetite.' In these cases the cause of the original dyspepsia has been removed, and the patient is suffering from a worse type of the disease induced by restricted dieting.

Many chronic cases require no medication, but most will be benefited by a short course of pepsin till the stomach is enabled to regain its tone. Papain as a routine is, however, preferable, since it can be given with full doses of alkali, in which medium its activity is not in any way impaired. The relief of the separate symptoms, and the general medicinal treatment of the various forms of dyspepsia caused by disturbed innervation, will be referred to in the following article.

DYSPEPSIA THE RESULT OF GASTRIC NEUROSIS

This is known as Nervous Dyspepsia, and, as implied in its title, it is a functional disturbance, the result of altered or disturbed innervation.

Etiology.—The condition is generally a part of a constitutional neurotic state, congenital or acquired—hence it is often associated with neurasthenia and hysteria; but in some cases it may be but the result of a reflex disturbance elsewhere, as is rendered probable by the well-recognised variety of vomiting or gastric disturbance associated with pregnancy and morbid conditions inside the skull. In other cases it may be the result of a poison manufactured somewhere in

the body, which selects for its action the gastric nerve-apparatus.

A gastric neurosis may show itself in various forms, according to the particular section of the gastric nervous mechanism involved; and unfortunately the subject has been greatly confused by the modern tendency to elevate every prominent symptom into a special type or variety of nervous dyspepsia. This is seen amongst the followers of Leube and Riegel, who sometimes are tempted to regard the most prominent symptom present as a distinct disease.

Any section of the gastric nervous mechanism may be the seat of disturbance or perverted action—viz. that which is responsible for the function of sensation, secretion, or peristaltic movement. Hence we have the following types of gastric neuroses: (1) *Sensory*; (2) *Secretory*; and (3) *Neuro-motor*.

The average case of nervous dyspepsia met with in daily practice presents a tangled web of symptoms, in which the separate threads of these three gastric neuroses are intricately blended, the most prominent in the mind of the patient being usually the sensory.

But other cases occur in which any one of these neuroses may be present in such an aggravated form as to overshadow the others; these will be referred to subsequently.

General Symptoms.—The ordinary nervous dyspeptic subject presents symptoms of gastric indigestion very much like those already detailed under 'Chronic Dyspepsia.' Thus at a variable period, generally about two hours after food, there is pain in the region of the stomach of the true gastralgic type; distension; a marked sense of oppression and gastric discomfort, which, in this variety of dyspepsia, generally passes into distinct distress; pyrosis, acid eructations, flatulency, nausea, retching or vomiting, and variability of appetite from bulimia to anorexia nervosa. The nervous symptoms are, however, out of all proportion more prominent and severe than in the simple chronic dyspepsia caused by irregular living, over-eating, indigestible food, improper mastication, &c. There is generally severe mental depression, over-anxiety about the trivial matters of life, and a tendency to hypochondriasis. Other evidences of the neurotic diathesis are forthcoming on examination of the family history and an investigation of the nervous system, and these are of considerable importance in the diagnosis. The contents of the stomach, if drawn off by the syphon, may be found normal and the hydrochloric acid in proper quantity, with very little mucus or excess of organic acid. Passage of the tube may reveal the stomach to be empty several hours after a meal, though the patient may be suffering from gastric depression and uneasiness, in which case food may be found to cause relief.

Since, as already stated, the three different forms of gastric neuroses may exist separately, it will be necessary to give a brief description of each.

1. SENSORY NEUROSES

The most important feature in this form of stomach-trouble is *Gastralgia*; it may be in rare cases the only symptom present, and hence it has been regarded by some writers as a distinct disease. Allbutt, in his classic article on 'Neuroses of the Stomach,' speaks of this pain in its typical form as a neuralgia of the stomach. It is paroxysmal; often, but not always, it is relieved by food; but its broad characteristic feature is that it comes on independent of food, sometimes in the middle of the night and sometimes in the morning before the first meal. In character, site, intensity, and duration, it varies considerably; it is of a burning, gnawing, or lancinating character, commencing in the epigastrium, sometimes extending towards the spine, but generally radiating from the pit of the stomach round the left thoracic margin towards the angle of the scapula. In its intensity it may amount to an agony as acute as that experienced in the passage of a gall-stone, and it may last from a few minutes to several hours without vomiting or other gastric symptom supervening.

Some tenderness is present, but this is always more or less diffused, contrasting markedly with the circumscribed tenderness elicited in gastric ulcer. Notwithstanding this tenderness or hyperæsthesia, the patient generally experiences some relief by pressing the epigastrium with his flattened-out hand, or by the application of a pillow. If he be in bed he may coil himself up or turn upon his face; and if in the erect position he bends his body forwards, and he has difficulty in straightening himself, owing to abdominal cramp. In very chronic and inveterate cases his trunk may tend to remain in this set condition when at rest. During a severe attack, the face is pale and anxious, the pulse quick, and the extremities cold, so that it may be mistaken for pseudo-angina.

Diagnosis.—This may be difficult, especially when the gastralgia is attended with other gastric symptoms; for it must be borne in mind that gastralgia may be sometimes caused by a *gastric ulcer*, by *cancer*, *hyperchlorhydria*, *organic acidity*, *acute dyspepsia*, or *gastro-succorrhœa*, and it may be simulated by *renal*, *hepatic*, or *simple colic*, and the gastric crises of *ataxia*. These different affections are eliminated by an investigation of the history and an examination of the gastric contents, which in simple or pure gastralgia are generally normal. But the most important diagnostic point is the presence of symptoms of hysteria or neurasthenia, and the aggravation or recurrence of the paroxysms about the menstrual periods or their occurrence during the menopause. In men

the writer has seen the most typical uncomplicated cases far more frequently amongst Jews than in other races.

Gastric hyperæsthesia is one of the most common symptoms of the sensory neuroses. It may be present in cases where gastrodynia never appears. The patient during digestion suffers from a sense of fulness, weight, or oppression in the stomach, always of a painful character, which may be described as gnawing, or burning, like the sensations caused in healthy subjects by eating very indigestible foods. More or less tenderness is generally present; and these symptoms may exist when the stomach is empty, or they may be brought on at any time by eating certain articles of diet which are usually harmless to healthy people, so that the neurotic patient becomes afraid to eat and markedly emaciates.

The diagnosis of the condition is usually easy, being always associated with neurasthenia or hysteria. Organic affections can be excluded by an examination of the gastric contents, which are found to be generally normal.

Perversions of the hunger-sense or *Anomalies of appetite* constitute a group of symptoms which are characteristic of the gastric sensory neuroses. *Anorexia nervosa* is the term employed to designate the almost total absence of the natural sensation of hunger—a condition met with in hysteria and neurasthenia.

Bulimia, the opposite state, is often witnessed, in which the appetite shows paroxysms of voracious activity; when this symptom is continuous it is styled *polyphagia*. *Akoria*, or the absence of the sensation of being satisfied or satiated, is often associated with bulimia; but it may exist without the latter in a patient who does not give way to gluttonous feeding, and who merely complains of feeling an empty void at all times in the stomach.

Pica, or the craving for indigestible substances which are not foodstuffs, is rare, except in patients who are insane; and the same is true of *coprophagy*, or the craving for filthy substances. Both perversions of appetite are met with in hysteria, but it is very doubtful if they should be classed as gastric neuroses.

2. SECRETORY NEUROSES

These occur in several well-marked forms, constituting the group of dyspepsias called by Campanella '*chemical*.' The varieties are known by the titles Hyperchlorhydria, Hypersecretion or Gastro-succorrhœa, Hypochlorhydria or Achlorhydria, and possibly Organic Hyperacidity.

Hyperchlorhydria.—This is the most important of the secretory neuroses. As its name implies, the condition consists in the secretion during the digestive process of a gastric juice containing an abnormally high amount of hydrochloric acid. The symptoms develop gradually. When the condition

is established there are in mild cases sensations of discomfort and weight about the stomach-region, coming on about two or three hours after a meal. In severe cases these pass into a burning or scalding pain accompanied by acid eructations and pyrosis, which may last for two or three hours, unless cut short by vomiting, which generally brings speedy relief. The condition is relieved by an albuminous diet and aggravated by starchy foods, and the pain can often be lessened by eating meat or egg-albumin, when the symptoms are acute, three, four, or five hours after a meal. Headache and giddiness are common symptoms. The disease is most frequently met with in chlorotic girls and young neurotic adults, and is usually associated with obstinate constipation. The tongue is clean, and the amount of flatulence always less than in cases of hyperacidity caused by the organic acids, because fermentation is absent in hyperchlorhydria. The nutrition and appetite are little interfered with, though the patient may be afraid to eat. The total acidity of the urine is always somewhat reduced during active digestion, but in hyperchlorhydria the urine may often be found to be alkaline and deficient in chlorides. A feature of some diagnostic importance is the relief afforded by a large dose of alkali.

Benedict's effervescence-test is applied by administering a teaspoonful of bicarbonate of sodium in a tumblerful of water an hour after an Ewald test-breakfast; if much acidity is present great bubbling or fizzling sounds may be heard on auscultating the stomach, and markedly increased tympanitis occurs. This procedure, however, gives similar results if the hyperacidity be caused by organic acids.

The only reliable test is to be found in an examination of the gastric contents, and this when possible should always be made.

The patient in the morning takes the Ewald test-breakfast, which consists of 1 to 2 oz. of bread and a half-pint of weak tea or water. In sixty to ninety minutes afterwards the soft rubber tube is passed, and the gastric contents withdrawn. The best reagent for all purposes to demonstrate the presence of free HCl is the ·5 per cent. alcoholic solution of *Dimethyl-amido-azobenzol*, which strikes a bright red colour when a few drops are added to the chyme, which generally need not be filtered. The depth or richness of the colour to the experienced eye is a fair indication of the amount of the acid present. The actual amount may be determined by filtering the coloured liquid and neutralising it by a solution of caustic soda till the colour disappears. A calculation of the amount of alkali used will give the percentage of free HCl present. In health this varies from ·1 to ·2 per cent., and is always greatly increased in hyperchlorhydria. The

total acidity, which is normally under 65 or 70, may rise to double this amount.

It must always be remembered that this condition is present in the majority of cases of ulcer, but the presence of the other characteristic symptoms and signs of ulcer are usually sufficiently in evidence to obviate errors in diagnosis.

The duration of the affection is variable, and is much influenced by mental over-work and worry, and by the presence of the emotional disturbances common in neurasthenia or hysteria. It is seldom continuous, being, except in the most severe cases, marked by intermissions. The prognosis is generally good, but must to a large extent depend upon the severity or intensity of the accompanying type of neurasthenia.

Hypersecretion, or *Gastrosuccorrhœa.*—In this neurosis there is an abnormal amount of acid gastric juice secreted by the empty or fasting stomach, independent of food. Two forms are met with—the *continuous* or *chronic*, which is sometimes called Reichmann's disease, and the *periodic* or *intermittent*. The intermittent begins suddenly with gastric discomfort, resembling that met with in hyperchlorhydria, which changes into an intense burning pain, soon to be followed by repeated vomiting of acid, irritating, gastric juice in very large quantities, which burns the throat and gullet. The attack, which may last for forty-eight hours or more, begins generally in the middle of the night, like acute dyspepsia, but the vomit contains no food, and may be bile-stained ; like acute dyspepsia, it may pass away as suddenly as it came. There is often severe headache and depression, with coldness of the extremities. This condition was described by Rossbach as *gastroxynsis* ; it is one of the rarest forms of gastric neurosis, and is with difficulty differentiated from the gastric crises of ataxia and some latent forms of gastric ulcer. An important point is the presence of neurasthenic symptoms.

The *continuous* form, into which the periodic may sometimes pass, begins like hyperchlorhydria ; the pain increases in severity till the end of digestion, and resembles severe colic, probably caused by spasmodic closure of the irritated pylorus. There is much hyperæsthesia, some gastric dilatation, and considerable tympanitis, which is relieved by the discharge of a large amount of acid gastric juice and some flatus. The absence of undigested food and organic acids exclude chronic dilatation caused by structural stenosis of the pylorus. Hypersecretion is a constant feature in some cases of ulcer, but the distinguishing feature of the neurosis under consideration is that large amounts of acid gastric juice can be syphoned off from the stomach *during fasting*, and the appetite may be abnormally increased, notwithstanding the gastric distress.

Hypochlorhydria.—This is a common form of secretory neurosis ; as its name implies, there is diminution of free

HCl in the gastric juice. When this is entirely absent *achlorhydria* is said to exist ; and when the normal ferments are also absent the condition is known as *achylia gastrica*. Though these conditions are frequently found in cancer, atrophy of the stomach, and chronic gastritis, they may unquestionably occur as a pure neurosis. As an example of the variability of the symptoms of the affection, the writer has had one typical case under observation for months in a lady of thirty years of age, whose chief complaint was an *intolerable sensation* of *gastric acidity*, which was constantly present with some precordial distress. The gastric contents, repeatedly examined by himself and carefully tested by Dr. Allen, failed to show a vestige of free HCl, and only the merest traces of combined acid, no organic acids, and but vestiges of the gastric ferments. The only neurotic manifestation was suppressed menstruation. Complete recovery followed a three months' course of HCl, and pepsin with strychnine ; an addition of about 15 per cent. to the body-weight ensued.

As a rule, the symptoms are those of severe indigestion, with much precordial distress caused by fermentation, which produces flatulence and vomiting of food, the latter but slightly altered. When the motor functions of the stomach are intact the symptoms may be almost nil, the case *quoad* digestion resembling the condition noticeable after a successful gastro-jejunostomy. Many cases formerly wrongly described as atonic dyspepsia belonged to this type of gastric neurosis.

(*Organic hyperacidity* occurs independently of structural alterations in the stomach, but it is doubtful if it should be included under the true gastric neuroses. Acetic, lactic, and butyric acids are found in various fermentative conditions of the gastric contents. Lactic acid is often present in cancer and in pyloric obstruction. The organic acids play a very prominent part in the chronic dyspepsia caused by errors in diet, and they are the chief cause of the severe distress and burning pain of acute dyspepsia.)

3. MOTOR NEUROSES

These consist of—

1. *Peristaltic unrest* — a condition characterised by loud gurgling sounds or borborygmi, which are increased by mental emotion, especially when occurring in the presence of bystanders, to whom they are sometimes painfully audible. Allbutt considers the condition to be indicative of motor weakness or delay, rather than of motor excess. They are always increased some time after a meal, and may extend to the intestines. Though generally associated with obvious hys-

terical or neurasthenic symptoms, they may be the only manifestations of the neurotic condition or of digestive disturbance.

2. *Nervous vomiting.*—In its typical form this occurs without apparent effort or previous nausea in hysterical women and neurotic children. The phenomenon may be brought about by the combined muscular mechanisms usually employed in ordinary vomiting, when the contents of the stomach are expelled by force through the open mouth ; but often it is caused by the unaided contraction of the stomach, as is usually the case in the vomiting of infants. In this latter case the food is simply regurgitated into the mouth and expectorated in small quantities by the patient. It may only occur at the beginning of the menstrual period. It is liable to be mistaken for cerebral vomiting ; in some instances it is a part of a gastric crisis in ataxia. The diagnostic characters are its association with hysteria and the absence of any marked deterioration in the physical condition of the patient. The writer has seen this neurosis diagnosed by two experienced London physicians in a case which the syphon proved to be one of almost complete pyloric obstruction from cicatrisation of an old ulcer, a portion of the food being regurgitated regularly three or four times a day. In this case the condition of the dilated bag of a stomach seemed to resemble that of the urinary bladder in prostatic obstruction, the overflow being regurgitated or vomited some time after each meal, which always consisted of liquids.

3. *Rumination* or *Merycism*, may occur. This is a rare condition in which the cud-chewing of ruminants is simulated, the food being regurgitated into the mouth, chewed again, and swallowed.

4. *Belching* is not uncommon, the gaseous eructations being forcibly and noisily expelled through the mouth. The nature of the flatus and the explanation of its source are described upon page 331. It is largely a question of habit, and should scarcely be regarded as a true gastric neurosis ; it may last for years and become incurable. In one well-marked case for years under the writer's observation the eructations were forcibly expelled whilst the lips were tightly stretched ; the resulting sounds resembled a series of loud trumpet-calls.

5. *Gastric spasm* is classed as a neurosis by Riegel and others. When both orifices become closed in wind-suckers or air-swallowers, the condition called *pneumatosis* occurs, the stomach being blown up by flatus so as to resemble the variety of meteorism caused by stricture at the splenic flexure of the colon. Spasm of the pylorus is generally a result of hypersecretion or of hyperchlorhydria, and is often seen in acute dyspepsia, where it is caused by the irritation of the

pylorus induced by butyric acid; it also occurs in gastric and duodenal ulcer.

6. *Gastric atony.*—Relaxation or motor insufficiency of the stomach is a part of every form of general debility and prolonged exhaustion resulting from acute or chronic disease. It is the condition which supervenes in chronic gastritis, and most cases of gastric disease from whatever cause, where a starvation diet has been adopted by the patient. By many authorities it is considered as a typical gastric neurosis independent of organic affection, and it may be regarded as corresponding to the true 'Atonic Dyspepsia' of old writers. The symptoms are those of delayed and disturbed digestion, with precordial distress and gastric pain associated with flatulence, much mental torpor, and sometimes signs of gastric dilatation. The diagnosis is made by the exclusion of organic disease and the introduction of the syphon-tube three hours after the Ewald test-breakfast, when food will be found in the atonic organ.

7. *Hypermotility*, or increase of the natural peristalsis or motor activity of the stomach, is the opposite condition to gastric atony. It is doubtful if such a condition should ever be regarded as a neurosis. Its existence has been assumed to explain those cases where the stomach is found to be empty soon after a meal. But the observations of Knapp go to prove that the explanation of this condition must be found in

8. *Pyloric insufficiency.*—In this condition, not only does the food pass at once through the pylorus, but the bile may regurgitate into the stomach. Knapp considers the pathology of the affection to be paralysis or muscular weakness of the pylorus following prolonged over-stimulation, and he finds it always yields to strychnine. The condition is diagnosed by the passage of the syphon-tube thirty or forty minutes after a test-breakfast, when the stomach will be found empty. An ordinary enema-apparatus being attached to the tube, air may be pumped into the stomach, when it will be found to pass onwards into the intestine, through the patulous pylorus. The presence of this neurosis may be in some cases a possible explanation of the condition termed by Boas *Gastralgokenosis*, which is characterised by a distressing painful sense of emptiness occurring within an hour or two after eating.

Diagnosis.—The special features of each of the gastric neuroses have been pointed out in the brief description of their signs and symptoms, and need not be here repeated, save to emphasise the great importance, and in many cases the absolute necessity, of administering a test-breakfast (that of Ewald answers all requirements) and in sixty to ninety minutes drawing off the chyme for analysis or examination. It is a safe rule never to arrive at the diagnosis of any gastric neurosis till

a scrupulous cross-examination as to the history, symptoms, and physical signs of the case warrant the exclusion of organic disease. Almost equally important is it to exclude those factors of common dyspepsia, as errors in cooking and eating, and the presence of foul necrosing molar teeth which may be interfering with gastric digestion in patients whose nervous gastric mechanism is normal. The diagnosis of any abnormal unrest or excitability of the nervous system supporting the suspicion of neurasthenia or hysteria is important, and when such is not forthcoming the influence of prolonged mental over-work, worry, or grief in originating or causing a return of the symptoms must be carefully weighed.

Prognosis.—This is as a rule satisfactory, but it must not be forgotten that many of the organic stomach-affections justify a more rosy prognosis than do the functional ones. The danger to life is practically nil, but the duration of the ailment may be exceedingly protracted; indeed, it may be fairly put in another way—that the prognosis of the gastric affection depends upon the prognosis of the general neurosis or neurasthenia of which it is but a local manifestation.

Treatment.—This should be *general*, directed to the abnormal condition of the nervous system; and *local* (using this term in a wide sense), directed towards the relief and cure of the gastric condition. Many neurotic patients show no signs of stomach-disturbance till mental fag, grief, worry, or excitement, irregularity in the time of meals, insomnia, &c., disturb their unstable balance; chlorosis may act in the same way. In these cases, when advice is sought for early, speedy relief of all symptoms may be fairly expected by a total change in the environment and habits of the patient, when such is possible, without drugs or the prescription of a fancy diet-list. Change of scene, and temporary rest from the worry of a harassing occupation by an enjoyable holiday, is the most reliable therapeutic agent in many cases.

Many of the cases of obstinate sensory neuroses, as *gastralgia*, *hyperæsthesia*, and *anorexia nervosa*, but especially the latter, will only yield to rigid isolation and a modified Weir-Mitchell treatment. In inveterate cases of so-called typical nervous dyspepsia, characterised by a blending of the symptoms of all the forms of gastric neurosis—secretory, sensory, and motor—where change of scene and rest fail, the Weir-Mitchell treatment with massage should be insisted upon. It is, moreover, the only treatment of any value in *nervous vomiting*.

Anæmia, constipation, and insomnia will call for appropriate remedies as urgently as do irregularities in meals, unsuitable and badly cooked food, imperfect mastication, severe mental or physical activity immediately before or after meals, &c.

As regards dietary, in addition to the remarks made under 'Chronic Dyspepsia,' it should be remembered that starchy

foods as a rule should be restricted in hyperchlorhydria, where they are very slowly digested, whilst albuminoids, or a strictly meat diet, may be freely given ; but in atonic and fermentative conditions albuminoids should be used very sparingly.

Some authorities, notably Fischl and Walko, recommend a diet of fats or oil in hyperchlorhydria and hypersecretion. Olive oil and cream in large amounts lessen the production of HCl, and hasten at the same time the digestion of albumins ; when such a diet is tolerated no other treatment may be necessary. Broadbent, upon the same lines, recommended a diet consisting largely of cream, butter, and buttered eggs. One great advantage possessed by the oil-therapy is that it can be freely used in those cases where the possibility of an ulcer is before the mind of the physician. Rectal feeding may be employed in many of the gastric neuroses, and the writer strongly recommends enemata of cod-liver oil. In achylia gastrica, peptonised foods are useful. In all the group of the gastric neuroses, tea, coffee, alcohol, and tobacco should be withheld or used very sparingly.

Drugs are indicated for the relief of the local symptoms. Gastralgia, if severe, may demand morphia, but this should be withheld when the pain is tolerable. Trousseau's plan of administering morphia in minute doses for its local anodyne action on the stomach is very valuable in most cases of gastric disturbance, and there is no risk of a habit being formed, as the drug should not be given in doses exceeding $\frac{1}{16}$ gr. ; it can be combined advantageously with other remedies.

A full dose (40 grs.) of bicarbonate of sodium may give relief ; oil of peppermint or menthol, belladonna, cocaine, and cannabis may be tried ; antipyrin is not usually successful. In chronic gastralgia and hyperæsthesia two drugs are of much service—viz. arsenic and silver nitrate ; they may be given in alternate courses in pill form. Hyperacidity must be met by large doses of alkalies given a few hours after food. Less than 30 grs. each of sodium and magnesium carbonate are useless ; bismuth and cerium salts are disappointing. Lavage, or thorough washing-out of the stomach, is valuable in severe cases. Hypersecretion yields to similar treatment, and lavage here may, as in hyperchlorhydria, be carried out with advantage by means of weak alkaline solutions. Much damage may be done in these cases, indeed in all gastric diseases, by dilating the stomach in the process of washing-out ; not more than 20 oz. liquid should be put into the stomach before depressing the syphon, and this amount may be repeated many times during the sitting.

Hypochlorhydria is generally markedly benefited by HCl in combination with pepsin and strychnine ; and where fermentation complicates the case papain may be occasionally substituted *for a short time*, given with alkalies and creosote.

Achylia gastrica is treated upon the same lines, but when severe all foods must be predigested by pancreatic ferments.

The motor neuroses are more difficult to treat. Thus nervous vomiting may demand Weir-Mitchell treatment, as already mentioned. Atony requires patient and persevering management, especially in the hours of feeding, which should not be separated by intervals longer than two hours, so that the bulk of food at any one time in the stomach is reduced to the smallest dimensions; and liquids for the same reason must be restricted, as in the treatment of gastric dilatation. Massage of the stomach, electricity to the abdomen, and vibratory stimulation are all beneficial. A minute perule containing $\frac{1}{16}$ gr. strychnine *ter die* is of great service in most cases of atony; lavage must be performed with care, only a small amount (10 oz.) of fluid being poured in at one time. Pyloric incompetence is benefited by strychnine and local massage. Nervous eructations and spasm of the stomach may be cured in most instances by convincing the patient of the evils of voluntary belching and of saliva-swallowing; and sometimes the disagreeable peristaltic unrest may be relieved in the same way. For this latter, strapping of the abdomen firmly with adhesive plaster, or the application of a firm abdominal belt with buckles, is the best routine, along with measures for the neurasthenic condition.

DYSPHAGIA

This, which simply means a difficulty in the act of swallowing, is a symptom of several diseased conditions; it may be the result of mechanical obstruction, spasm, pain, or paralysis, as in the following:

1. Inflammation of the tongue or malignant growths in the organ.
2. Inflammatory affections of the pharynx, tonsils, or larynx.
3. Ulceration, tubercular, malignant, or syphilitic, in the same sites, or the obstruction caused by retropharyngeal suppuration or growths.
4. Parotitis renders swallowing both painful and difficult.
5. Paralysis of the muscles of deglutition, as is seen after diphtheria and in chronic affections of the bulb—glossopharyngeal paralysis and tumours in the medulla—in the late stages of progressive muscular atrophy and general paralysis of the insane, and in acute bulbar paralysis the result of hæmorrhage, thrombosis, embolism, or acute inflammation of the medulla.
6. Œsophageal obstruction caused by (*a*) cancer; (*b*) stricture from narrowing, the result of corrosives; (*c*) the pressure of aneurismal growth, malignant tumours, and pericardial

effusion, thyroid enlargements, or adenomatous growths, and abscess arising in the bodies of the vertebræ ; (*d*) impaction of foreign bodies ; (*e*) inflammation—œsophagitis ; (*f*) paralysis, as after diphtheria.

7. Functional dysphagia is the result of spasm, which may have its site in any part of the tract, as in the palate, upper part of the pharynx, larynx, or œsophagus—a condition met with in hydrophobia and hysteria. When the spasm is limited to the gullet it is known as œsophagismus, a not very rare manifestation of hysteria and hypochondriasis.

DYSPNŒA

This is the name given to difficulty in breathing from whatever cause. The main element in the condition is the state of the blood, which contains a deficiency of oxygen or an increase of carbonic acid. It may contain an excess of the products of muscular metabolism, a condition which results in the dyspnœa of exercise. Poisonous products circulating in the blood of the medulla produce the dyspnœa witnessed in some cases of uræmia, diabetes, and fever. Dyspnœa is sometimes seen as a part of the psychic phenomena of hysteria, and as the result of irritation of the vagi from the pressure of tumours or aneurisms.

Where the deficiency of hæmoglobin, as in all forms of anæmia and chlorosis, interferes with the carriage of oxygen, and where oxygen is deficient in supply, as in poisoning by carbonic acid and in mountain-sickness and ballooning, dyspnœa results.

The following types of dyspnœa, resulting from abnormal conditions of the respiratory apparatus, are met with :

Inspiratory or *obstructive dyspnœa* is caused by obstruction to the air-inlet in the mouth, nose, pharynx, larynx, trachea, or bronchi.

The symptoms will vary with the seat of the obstruction. The number of respirations may be diminished, but the breathing is always laboured, and the difficulty as a rule is most marked during the inspiratory act. In blocking by tonsillar enlargement or adenoid growths, the noisy snoring character of the respiratory sound and the nasal tone of the voice are characteristic, and swallowing is difficult, especially in the enlargement from acute tonsillitis.

Laryngeal obstruction is easily recognised by the inspiratory stridor ; it may result from œdema, inflammatory swelling, false membrane, paralysis of the abductors, spasm of the adductors, tumour, or the presence of a foreign body. There is recession of the epigastrium, and deepening of the supra-clavicular hollows and intercostal spaces in the laboured

inspiratory act, with comparatively easy expiration; this is seen in croup and diphtheria constantly, and there is much laryngeal movement. Obstruction in the trachea or bronchi causes a dyspnœa of a less noisy character; the chest-movements may be unequal on the different sides of the thorax when one bronchus is blocked, and the auscultatory examination will corroborate this. Cough will generally be present, which is diagnostic.

Aneurismal tumours, malignant growths in the chest, and enlargements of the mediastinal glands, or abscesses from these latter or from spinal caries, are the usual causes of blockage of the main air-tubes. Inspiratory dyspnœa is also well marked in plastic bronchitis.

Expiratory dyspnœa is seen in its typical form in asthma and advanced pulmonary emphysema, in which conditions it reaches the degree of *orthopnœa*, when the patient is compelled to maintain the sitting posture, with great cyanosis and venous congestion, the expiratory muscles of respiration being all in violent action, though inspiration is comparatively easy. Bronchitis, with excessive secretion of a viscid nature, will produce this form.

Paroxysmal dyspnœa may be of the inspiratory type as in laryngeal spasm, or of the expiratory type as in asthma, or both inspiration and expiration may be greatly laboured as in cardiac dyspnœa and in uræmia. It is often a feature in the progress of thoracic aneurisms or tumours which press upon the pneumogastric nerve or its branches.

Exertion-dyspnœa, usually known as 'shortness of breath,' is seen after exertion or muscular exercise in cases of anæmia, debility, obesity, fatty heart, failing compensation, chronic bronchitis, emphysema, and all diseases in which there is damage of lung-substance, as phthisis and pneumonia. It exists in pleural and pericardial effusions and pneumothorax, and in abdominal diseases which tend to press the diaphragm upwards.

In all these conditions where the cause is sufficient to interfere with the aëration of the blood in the lung, the dyspnœa will tend to become marked during rest, and finally it remains permanent or constant.

Cheyne-Stokes respiration is sometimes classed as a variety of dyspnœa. In this condition the respirations at intervals become more and more rapid for a short period, after which they become slower and slower, till they finally cease in a dead standstill for a variable period of many seconds, after which the phenomenon begins anew. It is a grave symptom in most cases, indicating exhaustion of the respiratory centre, and is often followed by death in a few hours. These alternating periods of very rapid and of very slow breathing are often found in association with cerebral hæmorrhage, sunstroke, and uræmia.

ECTHYMA. (See under IMPETIGO.)

ECZEMA

This is a superficial inflammation of the skin associated with considerable itching and burning, and usually characterised by the exudation of a highly coagulable liquid plasma and the presence of a multiform eruption. It is impossible to give in any definition the distinctive characters of a disease so variable in its characters.

In the earliest stage the classic signs and symptoms of acute inflammation may be noticed—*i.e.* pain, heat, redness, and swelling ; the exudation in many instances, when observed soon after this, may be found in numerous vesicles which form from papules over the papillæ of the skin. The rupture of these vesicles leads to the escape of the serum, which continues to ooze or weep from numerous points. The presence of vesicles constitutes the most typical feature in the lesions of eczema. In some cases the eruption may not extend beyond the erythematous or papular stage, whilst in others the vesicles may become converted into pustules, but sometimes they are altogether absent ; as the disease progresses towards recovery scales are formed. All these conditions may be but stages or phases of a single attack, or they may all be visible at the same time, or only one or more of them may be present in any case throughout its course. Crocker divides eczema into four primary forms, according to the nature of the eruption, whether it be *vesicular*, *pustular*, *papular*, or *erythematous*, associated with more or less scaliness, all of which he regards as merely different expressions of the same morbid process. It is perhaps to be regretted that he did not make the *squamous*, or scaly, into a fifth primary form ; but he includes this under the erythematous as a sub-variety.

Etiology.—Eczema may appear at any age and at any part of the cutaneous surface. Some authorities exclude all cases of dermatitis which are caused by the application of irritants, and maintain that only those cases should be regarded as eczema which have an internal causation ; this is obviously an error, as probably external causes play a part in every case of eczema. The main element in the etiology of the disease lies in a peculiar vulnerability of the skin, probably of congenital origin. Individuals with this susceptibility develop eczema from trivial sources of irritation operating from the outside or inside. Thus cold or heat, friction, or the application of chemical substances, will induce an attack. Altered innervation, as in neurotic ailments and uterine disturbances, dyspepsia, constipation, Bright's disease, gout,

and all states associated with the retention of excrementitious products in the blood, will also act as exciting causes in those with skins susceptible to eczema. The writer recalls the example of a family, in six members of which a typical vesicular eczema would occasionally break out upon the back of the right hand after each had passed adult age: in one it would follow the use of strong alkaline soaps, in another an attack of acute dyspepsia, whilst others would only suffer after cold or exposure to east winds; rarely did the disease show itself in other parts unless upon the opposite hand. In all cases local treatment caused its removal. The theory of a common bacterial infection was untenable, since the condition was known to exist in those members who had settled in different climates. The old view still held by many recognises eczema as the outward manifestation of a *special diathesis* whose chief feature consists in a sluggish state of the general nutrition. If we regard one of the evidences of this sluggishness to be the above-mentioned vulnerability of the skin, the etiology of eczema ceases to be a puzzle.

Pathology.—Three theories are advanced to explain the catarrhal inflammation of the superficial layer of the skin which is the recognised process at work in eczema. These are that it is caused (1) by toxins produced within the body; (2) by altered innervation of the skin; and (3) by parasites. The latter view is supported by Unna, who describes a coccus which he finds growing in mulberry-like clusters in the epidermis; to this he gives the name of the morococcus, and he regards it as the origin of eczema, which is simply, according to his view, the result of the irritation produced by the toxin manufactured by the parasite. That parasites play the chief part in the multiform changes which occur in the eruption cannot be doubted; but the almost universal view is that these are secondary changes grafted upon the original catarrhal inflammation by the infection of the sterile vesicles after rupture. The chief agents in infection are the staphylococcus and streptococcus, which convert the vesicular into a pustular eruption. Seborrhœa is classified by Unna as a form of eczema (see 'Seborrhœa'), and it undoubtedly is parasitic; but it seems very probable that a true eczema may arise in the seborrhœic area and extend to other parts of the skin.

The anatomical changes in eczema are primarily confined to the papillary layer of the skin, the epidermis being affected afterwards. The lesions show swelling of the papillæ, dilatation of the blood-vessels, proliferation of the connective-tissue corpuscles, and exfoliation of the epidermis.

Symptoms.—These vary enormously with the stage of the disease, the intensity of the catarrhal inflammation—whether it be acute or chronic—with the region of the skin invaded,

and with the predominating features of the eruption—whether dry or moist, vesicular, papular, pustular, or erythematous ; hence endless varieties are described and a complex nomenclature obscures the entire subject of eczema.

About one-third of all the affections of the skin met with in patients of either sex and of all ages are eczema. Though the forms of the disease which are the result of a local irritation may remain confined to the seat of the irritation, as in the so-called ' trade ' eczemas, there is a general tendency in the eruption to become symmetrical.

The clinical division of eczema into *acute* and *chronic* is the most convenient, using the term ' acute ' not necessarily in the sense of severe, but in the sense of being acute in its development. Though acute in its onset, the disease is of indefinite duration, and often passes into the chronic form ; but this latter form may be chronic from its onset, and may become acute at any period of its slow career. (A similar clinical division of Bright's disease into acute and chronic in the same sense as the terms are here used is found useful at the bedside.)

Acute eczema may begin in any of the forms accepted as primary varieties by Crocker—*i.e.* vesicular, papular, pustular, or erythematous. Typical acute eczema commences with local symptoms ; rarely, except in very severe cases, are there any signs of fever, malaise, or other constitutional disturbance. The first local symptom is a sensation of burning heat and itchiness in some patch of skin ; coincidentally with this, or immediately after, redness and swelling appear. At this early stage the condition is practically an *erythema* ; and it may remain as such, especially when the face is attacked, and gradually it passes off in branny desquamation. In the acute typical disease under consideration, however, the erythema rapidly passes into a *papular* stage, which is so short as generally to be unnoticeable, the papules becoming vesicles within a few hours after the onset. The elevations being generally found to contain fluid from the moment of their first appearance, the papular stage in acute eczema is considered by many to be purely hypothetical, the *vesicular* stage following direct upon the erythematous. The point is of no clinical importance in the majority of cases of eczema, but it should be remembered that a form of eczema does begin and remain throughout its obstinate course as a true papular eruption ; this was formerly called lichen simplex. After a very short period—it may be a matter only of hours—the vesicles rupture either spontaneously or from scratching, and the stage of weeping or discharge is established.

This is the most characteristic of all the features of eczema : from the ruptured vesicles separate drops of a clear, sticky fluid exude which stain the patient's linen and stiffen it in

a characteristic manner. Little, if any, relief to the burning and itching follows the oozing, which may continue for a considerable period, successive crops of vesicles appearing and bursting ; or it may speedily dry up spontaneously, when the affected part will be covered over by crusts, or a slower and more gradual resolution may take place, when the affected skin will become indurated and dry, and covered over with scales somewhat resembling psoriasis. This, which may be tegarded as a frequent termination of the disease, somerimes exists from the beginning in chronic cases, when it is described as *scaly* eczema.

The progress of an otherwise typical acute eczema beginning with an erythematous stage may not pursue this regular course, but may show signs of pus, the vesicles in this case being probably infected by staphylococci or streptococci. In this stage the eczema is called eczema *impetiginoides* ; but in cachectic subjects it may take on the pustular form from the onset, as on the heads of strumous children the pus on drying up forms dark-brown crusts or scabs, underneath which a free suppurating surface is found.

Chronic eczema may result from an acute attack, or it may be chronic from the beginning, appearing gradually in the form of papules, pustules, erythema, or vesicles, or of a mixture of all these forms. The most typical example of the condition is seen in the so-called eczema *rubrans*, eczema *madidans*, or weeping eczema, seen so often in the legs of old people, which may begin in the vesicular or pustular form. The entire limb becomes a deep dusky red, and has a raw appearance from the destruction of the epithelium. A copious discharge of clear amber or colourless plasma oozes from the raw surface. As this dries up, a continuous crust is formed, underneath which the exudation is constantly going on ; blood is sometimes mixed with the discharge, and ulcers often form, especially when varicose veins are present. After a very long and tedious course, the surface of the eczematous area may become squamous, or scaly.

Dry, squamous, or *scaly eczema* is a common form of the chronic disease. Though many cases of acute eczema wind up in this fashion, many others begin and end in this type without exhibiting any exudation unless when irritated or scratched. These dry scaly patches resemble psoriasis, and have a thick indurated base, but the scales are branny and do not cohere into plaques ; they may remain for years on the neck or limbs.

Dry papular eczema was formerly described as lichen simplex ; it is seen in patches on the back and extensor surface of the limbs. The papules are red and generally discrete, about the size of a millet-seed, and give rise to intolerable tching ; when scratched, the papule may be seen to contain

a little point of fluid, and often bloody crusts form from injury produced by the finger-nails.

The natural tendency of the dry chronic form is towards the formation of much induration and hypertrophy, and hence it has been described as *eczema sclerosum* or *rimosum* ; fissures may form, as are seen in the thickened masses of epithelium on the palms and soles. *Warty* or *verrucose* eczema is the name given to the form accompanied by hypertrophy of the papillæ.

The local varieties of eczema are numerous, and need not be described. They all possess the features, pure or mixed, of the types already discussed. In the palms of the hand the growth of epithelium becomes so great as to mask the true nature of the disease, and the nails are much split up or lamellated. Owing to the board-like rigidity of the epithelium it splits up, causing fissures which extend down to the corium ; these become very painful and often bleed. The condition is a true eczema, but from its appearance it is sometimes described as *psoriasis palmæ*.

When eczema affects the flexures it generally assumes the moist vesicular form, whilst the extensor regions are usually the seats of the dry forms. The scalp and forehead often show the purulent type, especially in children, whose eczemas always tend to become purulent or impetiginous ; whilst in the aged the tendency, except in eczema rubrum, is towards the scaly form. Eczema affecting the anus and genitals is most obstinate and distressing, the itch being intolerable ; in these localities it may begin and remain as the erythematous type. A variety of eczema affecting the hair-follicles on the extensor surfaces of the arms and legs is described by Malcolm Morris as *follicular* eczema, each inflamed follicle standing out as an angry-looking papule ; these congregate into intolerably itchy patches. He considers this form of parasitic origin.

Diagnosis. — This is extremely difficult to the inexperienced, but the difficulties pass rapidly away after observing a few cases closely from onset to finish. There is usually little doubt in all cases of moist eczema exhibiting a *continuous* sticky secretion which stiffens and stains the patient's linen. The difficulty in diagnosis is greatest in the dry varieties of the disease.

Psoriasis never shows any secretion. Its usual site is on the extensor surfaces of perfectly healthy-looking subjects. The patches have very well defined margins, which do not shade off into the normal skin ; the plaques consist entirely of silvery scales which, when forcibly removed, show minute bleeding points, but never a weeping serous discharge. On the scalp the chief diagnostic point is the abruptly circumscribed margin and the absence of all evidence of inflammatory exudation in the crusts.

Pityriasis rubra comes on suddenly, and involves the entire surface of the body. There is little burning or itching, and never amber crusts ; any exudation which may be present does not stain and stiffen linen as the secretion of eczema does.

Lichen planus exhibits flat, shining, discrete, purplish papules showing a central horny point occurring commonly on the wrists or inside of the thighs, and never discharging fluid or forming crusts.

Urticaria, especially of the papular variety, is recognised by the scabs which form on the top of the pale red papules after scratching ; it is usually seen upon the loins or buttocks of dirty children, and there is always a history of wheals.

Erythema intertrigo is only seen in fat patients or infants where two folds of skin are in constant contact, as in the groins or breasts, or folds of the neck or buttocks ; there is a copious secretion which stains but does not stiffen linen.

Tinea or *Ringworm* and *Favus* on the scalp are recognised, the former by the stumpy broken hairs, which are easily pulled out ; the latter by the cup-shaped yellow odorous crusts ; and the parasites of both are demonstrable by the microscope. On the body the sharply defined circular patches or rings of tinea can scarcely be mistaken.

Sycosis closely resembles chronic eczema of the hairy parts of the face, but it begins in the hair-follicles ; the onset and progress are much more rapid, and the hairs speedily become loose. The microscope reveals the fungus in the sheath.

Scabies is the condition which gives rise to the greatest difficulty in diagnosis, and a mistake is a serious matter, especially in schools. The difficulty is intensified by the fact that the irritation caused by the itch insect and the subsequent scratching produce a true eczematous eruption of papules, vesicles, and pustules, with more or less erythematous redness. These, however, do not occur in continuous patches, but in irregularly scattered islets, whose favourite sites are the front of the wrists, between the fingers, the inside of the thighs, the armpits, penis, and toes. Crocker states that a pustular eruption on the hands or feet of an infant is nine times out of ten due to scabies. If a careful search is made, the burrow will be found with its mouth marked by a vesicle, and the parasite may be impaled upon the point of a pin thrust through the epidermis into the blind end of the burrow.

Impetigo contagiosa closely resembles pustular eczema, and an ordinary eczema may readily be infested by the parasite, staphylococcus aureus. The crusts are yellow like honey, there is no continuous oozing as in moist eczema, and the eruption is not symmetrical. As its name implies, it is infective and contagious, the disease being spread by inoculation with the finger-nails. When it affects the scalp,

it is generally associated with the presence of pediculi. A notable feature is the swelling of the neighbouring lymphatic glands.

Prognosis.—It is doubtful if ever the disease shortens the duration of life, if we except those rare cases which pass into pityriasis rubra. Eczema is generally liable to relapses, and these are the rule in those cases whose family history shows an inherited vulnerability of the skin, and where the eliminatory functions are at fault, as in the gouty. Acute vesicular eczema, as a rule, speedily responds to treatment ; but the dry papular and palmar forms may prove most intractable, and in rare cases the eruption or scaly patches may defy treatment.

Treatment.—The important question crops up in most dissertations upon the treatment of eczema as to whether the first place should be given to constitutional or local measures. Those who regard the irritation as internal place their reliance upon constitutional measures, whilst those who consider the original irritation as coming from without depend solely upon local applications. Unquestionably local treatment is of primary importance, but alone it will often fail ; and the only sound practice is to avoid any routine method, and treat each case in the first instance after an exhaustive examination of the probable causes which have originated, or which are factors in keeping up, the diseased process.

Every departure from the laws of health should be corrected. Gastric troubles, anæmia, chronic constipation, the uric-acid diathesis, and faulty elimination of all kinds should be met by the recognised agents suitable in each case. Overwork, late hours, irregular meals, and harassing mental occupations, when possible, should be avoided.

Dietetic routine treatments, so popular with patients, had better be discountenanced, as these often tend to perpetuate the disease. In chronic cases a generous mixed diet prescribed upon sound lines is much to be preferred to a cut-and-dry unvarying dietary ; but any article of diet which the experience of the patient proves to be injurious must be forbidden. Thus, wines and beer are at once known to the patient to be injurious by the increased burning or discomfort experienced in the eczematous patches. This is well recognised by everyone who suffers from facial eczema, and it is well to insist upon the exclusion of alcohol. In special cases a little pure whisky well diluted may be permitted. Saccharine foods are, as a rule objectionable ; sugar, jams and jellies and sweet fruits, especially strawberries, must be avoided. Salt meats, pork, pastry, and cheese are objectionable.

The diet in acute eczema should be mainly milk and light farinaceous foods.

Of specific drug-treatment internally there is none. Arsenic, so highly praised by some, is positively injurious in acute

cases, but in chronic scaly eczema it is often most valuable. Antimony in small doses generally is very valuable in acute cases. The writer often obtains much benefit from a short course of chloride of calcium where there is much exudation in acute cases.

Crocker recommends the internal use of turpentine, and counter-irritation over the spine in cases marked by recurring exacerbations; but it must always be kept in mind that the irritated area may become the site of a fresh patch of the disease. Sulphur and ichthyol are advocated by some, but they often upset the digestive apparatus.

Local treatment should be employed in every case: the main indication in the acute disease is to shield the inflamed surface from every possible source of irritation. Though change of scene is a most valuable adjunct, there is danger of sea-air aggravating most cases of eczema; patients should not be sent to the coast or to places exposed to north and east winds. Water should be most sparingly applied, and the use of soap avoided in all acute cases. The skin in these cases may be cleansed by the application of a soft rag smeared with olive oil.

It is useless to commence the local treatment of any case of eczema without first removing all crusts, scales, and inflammatory products. This is most safely accomplished by the application of a bread-and-water poultice for two or three hours.

Local applications in the form of lotions, paints, ointments, pastes, powders, &c., which have proved efficacious are innumerable. The author, in his 'Dictionary of Treatment' (4th ed.), pp. 352–363, has given the formulæ for many of these, and here only the general principles which should guide the local treatment need be mentioned. The main consideration to be kept in mind is that agents which at one stage of the disease are curative, at another may be most injurious.

At the commencement of an acute eczema, or at any period whilst the inflammatory process is severe, bland unirritating emollient applications are alone admissible, and their use should be continuous, so as to protect the inflamed surface from the irritation caused by changes of temperature and from the introduction of septic organisms. A note of warning must, however, be raised in order that this method be not abused by permitting the applications to cake and form an impervious crust which causes the retention of the pent-up irritating secretion underneath; when this occurs the ointment or powder should be periodically removed by gentle friction with a soft fabric saturated with olive oil. When the discharge is *very* profuse a weak lotion of lead, containing zinc oxide in suspension, is highly efficacious, or the free dusting

of the part with prepared fuller's earth or zinc powder with starch and a little boric acid.

As a rule, the average case of acute eczema in child or adult with moderately profuse secretion can be easily treated by an ointment consisting of 1 part of prepared calamine to 7 of zinc ointment. Lassar's paste, consisting of 2 oz. vaseline, 1 oz. starch powder, 1 oz. oxide of zinc, and 40 grs. salicylic acid, is a good routine application smeared over the part or applied freely on lint.

Chronic eczema of the weeping type may be treated upon the same lines, but a more astringent application is usually necessary ; lead is useful, a drachm of the strong liquor being added to each ounce of zinc ointment. It is a good plan to commence the treatment of such cases, as if they were of recent origin, by emollients ; after a few days lead can be combined with the ointment, and tar can afterwards be very cautiously added, gradually increasing the proportion after watching closely its stimulating effect. Tar is, as a rule, contra-indicated in all acute cases, and if applied in chronic cases in a too concentrated strength it is very liable to convert them into the severe acute forms. In the chronic moist eczema of the legs, especially when associated with varicose veins, local applications are generally useless unless the patient be kept in the horizontal position or the limb be enveloped by an elastic bandage.

Chronic dry eczema can only be successfully treated by tar. It is a good plan to begin with a lotion of 1 part of the alcoholic solution in 8 of water, and after all scales have been softened by this to replace it by an ointment of the same strength with lanolin, to which 2 per cent. of white precipitate is added. Tar not only establishes an altered action in the diseased skin, but it effectually relieves the burning itch which tends to perpetuate the disease by producing scratching. It is needless to say that all patients should be warned against the evil of yielding to the desire to scratch the affected part, and children should be prevented from so doing by enveloping the hands in woollen gloves at night. Where pustules are a prominent feature, these should be got rid of by an antiseptic before applying tar. Saturated boric lotion is efficacious, but a 2 per cent. iodoform ointment is better. Palmar eczema is most intractable, and little progress can be made till the thickened epidermis is removed. This can be done by the daily application of a 10 per cent. salicylic-acid plaster muslin, after which a 15 per cent. tar ointment containing 2 per cent. salicylic acid may be used. The use of india-rubber gloves is indispensable in the treatment of the most chronic cases.

Scrotal and vulvar eczema give rise to much distress, and lotions are to be preferred to ointments. Lead may be freely

used in the presence of much secretion. It should be combined with calamine or zinc oxide. Crocker recommends occasional painting of the part with 5 grs. nitrate of silver dissolved in 1 oz. nitrous ether, and applying a mustard plaster over the lumbar region to control the itching. Anal eczema is best treated by conium ointment, diluted with an eighth part of liquor carbonis.

Eczema of the face can best be treated in children by Lassar's paste smeared over the face thickly, and covered by a lint mask. In adults, a good plan is to apply a lotion of calamine (1 in 50) as long as the patient is able to stay in the house: and when he must go out of doors it may be permitted to dry, when the excess of powder can be brushed off with a soft silken cloth. After the acute stage is over, a 1 in 16 ointment of liquor carbonis, with a little lead and white precipitate, may be continuously applied, the excess being gently rubbed off when the patient has got to appear in public.

The X-rays have been successfully employed in chronic cases, and Schmidt claims for this method excellent results in the chronic hyperkeratotic palmar variety and in the eczema of the anus, when all other agents had failed.

ELEPHANTIASIS. (See under CHYLURIA, p. 221).

EMBOLISM

When a detached thrombus, pieces of blood-clot, or of valvular vegetations, scraps of parenchymatous, atheromatous, or cancer tissue, bubbles of air, drops of fat, parasites, or foreign bodies free in the blood-stream become impacted in any arterial trunk or branch which is too narrow to permit their passage, embolism is said to have taken place, and the plugging substance is called an embolus.

Thrombi are the commonest source of emboli, and though these very often originate in the veins, the resulting emboli cannot be stopped when once they become free in the blood-stream till they pass through the heart and plug the pulmonary artery or its branches, or till they become arrested in the branches of the portal vein in the liver.

Pulmonary emboli are also formed by the vegetations detached from the valves and lining of the right auricle or ventricle.

It is obvious that embolism, if we exclude the portal system, is an arterial phenomenon as regards its site, since the veins increase in size as they approach the heart, and cannot be blocked by emboli detached from their smaller branches.

The blocking of the systemic arteries is caused by thrombi or vegetations detached from the left heart or pulmonary veins, or from some artery between the heart and the site of the plugging. In very rare cases emboli have been known to originate in the systemic veins and pass directly into the arterial system through a patent foramen ovale. Retrograde embolism is the name given to the rare occurrence where emboli are carried in the veins in the direction opposite to the blood-current. (See also under 'Thrombosis.')

When the plug is free from bacterial infection the results which follow its arrest are simply those of diminution of blood-supply, but if the occluded artery is rich in anastomotic branches no appreciable result follows, and the same thing occurs if the embolus happens to block only a portion of the lumen of the artery. Local anæmia often means the death of the part supplied by the occluded artery, as is seen in the gangrene of the limb following plugging of the femoral. Welch, in his exhaustive article in Allbutt's 'System,' points to the importance of recognising the differences observable in the various tissues and organs whose blood-supply is cut off, the more highly differentiated specific cells of an organ suffering first. Thus, the ganglion-cells of the brain die in less than half an hour after the stoppage of their arterial supply. This is the explanation of the rapidity with which softening and other changes follow cerebral embolism, because the arteries are *terminal*, there being little or no communication between the branches of neighbouring vessels, a condition which also exists in the retina, spinal cord, kidney, spleen, and coronary arteries.

The local anæmia may pass off, leaving no trace of injury to tissues or organs, or it may end in the sudden death or gangrene of the part or in the slower softening or withering-up of the elements, or the phenomenon known as *hæmorrhagic infarction* may occur. This consists in a hyperæmia and extravasation of blood in the area supplied by the occluded artery, the red corpuscles passing out by diapedesis through the capillary walls. When hæmorrhagic infarction occurs in the soft spongy tissue of the lung, and in the mucosa of the intestine, the infarcts form wedge-shaped masses of dark red colour and firm consistence, the apex of the wedge or pyramid corresponding to the embolus and the base extending towards the periphery. In the brain, such infarcts rapidly cause breaking up of the tissue, as in an ordinary apoplectic hæmorrhage. In the kidney and retina the infarcts are pale or white, whilst both varieties may be found together in the spleen.

These pyramidal-shaped masses undergo gradual absorption, and may leave nothing but slight scar-tissue behind them; in the brain, a pigmented cyst may be the only remnant of their previous existence.

When the embolus is caused by vegetations of bacterial origin, as in malignant endocarditis, to the above mechanical effects are added those produced by the specific poisons secreted by the infective virus. Inflammatory changes supervene, which often end in the formation of pus, and the establishment of pyæmia, or the bacteria may invade the arterial wall, and by destroying the vitality of the intima or media, an aneurism may result. These are more common in the brain, but may occur anywhere ; the exact site of the aneurism is not above the original site of the embolus, but corresponds to the region of the artery destroyed by the micro-organisms.

Air-embolism is met with during operations involving the opening of veins in the base of the neck, the air being aspirated through the wounded vessels during the inspiratory act ; it has also been observed to follow uterine injections, and is a feature in caisson-disease. Welch has discovered in the blood of patients succumbing to air-embolism an anaërobic bacillus which generates gas freely. The entrance of air into a vein during a surgical operation is sometimes demonstrated by an audible sound, followed almost immediately afterwards by violent symptoms of suffocation which may rapidly prove fatal ; but recovery has been many times recorded.

Fat-embolism is much less serious than air-embolism. It follows fractures and injury to bones containing fat-marrow, and has been noticed after vigorous passive movements for the relief of anchylosis of joints. The blocking is capillary, and is usually seen in the lung, brain, and kidney. The symptoms are increasing dyspnœa and cyanosis, with bloody frothy expectoration, and distension of the cardiac chambers ; when the brain-vessels are blocked, apoplectic symptoms supervene ; the renal form may show itself by the presence of oil-globules in the urine. As regards the possibility of fat-globules passing through the capillaries of the lung, the reader is referred to page 167.

Symptoms. — These vary with the locality of the occluded artery, and as a rule their onset is *sudden*. It must not be forgotten that many of the cases of emboli met with clinically are instances where a portion or the whole of a thrombus growing, say, upon the mitral valve, or upon a patch of atheroma in the aorta, becomes detached, and as it is swept onwards it reaches the bifurcation or branching part of some artery, which it is unable to block. In this situation the plug, technically called a *riding embolus*, remains, and becomes the nucleus of a secondary thrombus, whose extension may *gradually* occlude the branches of the vessel. In this way the slow ingravescent symptoms of a thrombus may follow those of incomplete embolism ; or a secondary embolus may

become suddenly detached from the secondary thrombus and cause blocking of a distal branch of the same artery.

Pulmonary embolism, the source of which must be a thrombus in some of the systemic veins, or in the right heart, may be instantly fatal from cardiac syncope when the embolus is very large. Usually the patient is suddenly seized with collapse, severe dyspnœa, and cyanosis, preceded by pallor; the action of the heart, at first tempestuous, in a few moments becomes weak and irregular, and the pulse soon disappears; profuse perspiration and dilatation of the pupil usher in syncope or convulsions. Examination of the lung will demonstrate nothing to account for the gasping or lividity or engorgement of the veins of the neck, the diagnosis being mainly formed upon the appalling suddenness of the dyspnœa and the presence of a thrombus known to have been present in some of the superficial veins or in the heart.

Where the smaller divisions of the pulmonary artery are blocked, hæmorrhagic infarcts form, and the symptoms may be slight or absent; there may, however, be physical signs of dulness and absence of the breathing over an area corresponding to the base of the pyramidal consolidation; bloody expectoration is common and very characteristic. Inflammatory signs of a localised pneumonia, with some pain and fever, are often present; coagulation-necrosis or pyæmic abscess may form if the patient survive.

Coronary embolism is rare; thrombosis is more common, especially in the angio-sclerotic heart. The symptoms are sudden cardiac distress and a rapidly fatal failure of the heart, accompanied by dyspnœa and angina-like symptoms, lasting but a few minutes.

Hepatic infarction may give rise to no symptoms, owing to the free anastomoses of the capillaries of the portal vein and the intra-hepatic branches of the hepatic artery. When the emboli are septic, pyæmic abscesses form.

Splenic embolism is usually latent, but large infarcts may cause sudden pain and splenic enlargement, with tenderness and a friction fremitus.

Renal embolism is often latent; but when large infarcts are formed there is severe pain with tenderness, and some blood appears in the urine. Abscesses form when the emboli are of septic origin.

Aortic embolism can only occur in the thoracic part by the detachment of a large fibrinous mass, or blood-clot, from an aneurismal sac, and this may block the region near the sinuses of Valsalva and cause sudden death. Embolism of the abdominal aorta is more common; the usual site is near to the bifurcation, but the embolus may extend upwards to the origin of the inferior mesenteric. The occlusion is probably thrombotic in most of these cases, as the plug always extends

downwards into the iliacs, the origin of the thrombus being an embolus which only partially blocks the vessel, as it is trapped at the bifurcation. Mitral stenosis, as pointed out by Welch, is not rarely associated with plugging of the abdominal aorta. The patient is seized with severe pain in one or both legs, followed by an increasing paraplegia, which may be complete in a few minutes or hours; sensation is lost sometimes as high as Poupart's ligament, and both limbs become rapidly pale and cold; the pallor is soon followed by a dusky mottling. Pulsation is absent in all the arteries of the limbs and in the iliacs. Death occurs in one to four days, but life may be prolonged for many days or weeks, in which case gangrene is almost certain to supervene in one or both limbs; bedsores, bladder-trouble, abdominal tympanitis, diarrhœa, and anasarca may complicate the condition. In a few cases, however, recovery has been reported.

Cerebral embolism is described upon page 167.

Femoral, *popliteal*, *axillary*, and other sites of arterial plugging are obviously more easily recognised than when the arteries of the trunk are the seat of embolism. There is sudden severe pain, loss of sensation and power in the affected region which becomes cold, and all pulsation below the plugging is absent. Gangrene is very liable to occur unless the anastomosis is very free. In a case recently seen by the writer, the femoral was found completely blocked a little below Poupart's ligament, pulsation being vigorous above, but entirely absent in the vessel below the embolus, in the posterior tibial, and in all the arteries of the limb; the leg and thigh were cold, and sensation was lost. The advent of gangrene seemed a certainty in this case, especially as the embolus was the result of acute ulcerative endocarditis; but within seven days the circulation was perfectly restored, an artery in the buttock, almost as thick as the little finger, being found carrying the blood to the limb: it was probably a branch of the sciatic or of the gluteal from the internal iliac; pulsation in the posterior tibial was normal. The patient—a physician—died some weeks afterwards from cardiac thrombosis or cerebral embolism.

Superior mesenteric embolism, when the larger branches of this artery are plugged by an embolus or thrombus, often leads to marked intestinal infarction, which may involve the bowel from below the duodenum to the transverse colon. The onset is sudden, with much colicky pain and abdominal tenderness, and some collapse; these symptoms are soon followed by loose, offensive, tarry motions, which are highly characteristic. Vomiting soon supervenes, with symptoms of general peritonitis and obstruction, and the case may be easily mistaken for one of perforative peritonitis, obstruction, or intussusception. Laparotomy and resection of several feet

of bowel have been followed by a successful issue in some cases. The diagnosis must mainly depend upon the presence of thrombi or emboli in the heart or large arteries, and upon the absence of the history or symptoms indicative of other intestinal lesions.

Prognosis.—This will depend upon the importance of the parts whose blood-supply is occluded, and upon the thoroughness with which the occlusion has taken place, embolism being more liable to be followed by local necrosis where the arteries are terminal. Cerebral embolism is very liable to be followed by the death of the highly organised ganglion-cells. The prognosis is always very grave where the emboli are laden with septic, or pus-forming organisms.

Treatment. — Little can be done beyond compelling the most absolute rest with the view of prevention of further detachment of thrombi; and where thrombosis is known to be present, as in an inflamed varicose vein, the importance of rest as a preventive of pulmonary embolism is of vital moment. Friction is always contra-indicated. When the artery of a limb is blocked, warmth should be applied and the part wrapped up in layers of wool without pressure. The only drug-treatment available is large doses of ammonia well diluted, to reduce the coagulability of the blood. The writer once injected diluted ammonia solution into the carotid with marked temporary benefit in cerebral embolism. In the embolism of malignant endocarditis sulphocarbolates should be pushed in full doses. In embolism or thrombosis of the superior mesenteric artery or vein, laparotomy and resection of bowel may save life.

EMPHYSEMA

This term in its general sense is employed to indicate the surgical condition in which air has found its way into the cellular tissues. The common cause is the puncture of the lung by the extremity of a fractured rib, or wounds of the trachea and bronchi, or rupture of the pulmonary vesicles during violent paroxysms of coughing and straining. This form is termed *interlobular* or *interstitial* emphysema, when the air escapes into the inter-alveolar tissue, and is a not rare occurrence after tracheotomy; it may produce pneumothorax and lead to collapse of the lung.

The condition will remain latent unless the air travels along the root of the lung and the trachea and invades the cellular tissue at the root of the neck, from which it may extend over the body. Injury to the nasal sinuses from fracture of the base of the skull may cause orbital emphysema;

œsophageal, gastric, and intestinal perforation may also cause a more or less general diffusion of air in the cellular tissues.

The part affected is swollen and soft, and to the touch yields the unmistakable sensation of air-crepitation.

Unless when pneumothorax occurs, the condition is seldom serious, the air becoming gradually absorbed. In post-tracheotomy emphysema, the air passes from the wound downwards, below the deep fascia, and, following the course of the bronchi, may appear in patches or bubbles under the visceral pleura ; it may pass backwards or forwards, invading the mediastina, or it may end in general emphysema of the neck, arms, or trunk.

Interstitial pulmonary emphysema has no pathological connection with the following, save in those rare cases where it arises from rupture of true emphysematous vesicles or bullæ under the pleura.

EMPHYSEMA OF THE LUNGS

This may be defined as a permanently dilated condition of the terminal inter-alveolar passages, accompanied by atrophic changes in the walls of the dilated air-cells.

The most common and typical variety of the disease is known as *Large-lunged* or *chronic hypertrophic emphysema.*

Etiology and Pathological Anatomy.—The disease may begin at any age. Hereditary or congenital weakness of the pulmonary tissue, especially of the elastic fibres which support the alveolar walls, is believed to be a powerful predisposing cause, as their absence or disappearance is a constant secondary result of the atrophic changes. Occupations which involve severe muscular effort and constant straining, as glass-blowing and playing on wind-instruments, are important factors. Diseases of the lungs or bronchial apparatus, or any condition which leads to prolonged or constant coughing, as bronchitis, whooping-cough, asthma, cardiac dyspnœa, chronic phthisis, &c., are liable to produce the affection.

Of the various theories propounded for the elucidation of the disease, that of Jenner is the most widely accepted. It is known as the *expiratory* hypothesis. According to this theory, the dilatation of the air-cells is caused by the increased pressure of the air in them during the moment of forced expiration in all straining efforts with a closed glottis, and in the act of coughing. The chest being full of air, and the glottis closed, the expiratory muscles when brought into action will compress the lung ; those parts of the pulmonary tissue which receive the least support from the chest-walls at the period of compression will suffer first and most severely. This is the obvious explanation of the advanced condition of

dilatation found in the apices, along the anterior margins of the upper lobes, and in the edges of the bases.

Other or all portions of the lung ultimately suffer if the heightened pressure within the air-vesicles is frequently repeated, as in the coughing of chronic bronchitis, in cornet-playing, glass-blowing, &c. The distension of the vesicles or air-cells leads to atrophy of the intervening septa, so that several vesicles coalesce, or are stretched into one, the elastic-tissue, epithelium and capillaries disappearing. Ultimately the distended, coalesced, and ruptured cells cause the lung to approach the reptilian or batrachian type. As the volume of the lung increases, the costal cartilages and sternum are pushed forwards, causing the chest to assume the barrel-shape so characteristic of the disease. Niemeyer maintained that when the glottis was closed the compressed air driven from the lower bronchi descended, owing to the oblique downward direction of the upper bronchi, and distended the air-cells connected with these latter tubes.

The *inspiratory* theory was promulgated by Laennec, who held that the heightened pressure was caused by the distending force of inspiration. The late Sir William Gairdner adopted this hypothesis in explanation of emphysema in chronic bronchitis and phthisis, which he believed caused collapse of air-vesicles, the neighbouring ones becoming distended by the pressure of the inspiratory wave of air (*compensatory* emphysema).

The supporters of the *atrophic* theory endeavour to explain the phenomenon of emphysema on the assumption of a primary failure in the nutrition of the alveolar walls, which leads to degenerative changes. That such nutritive or degenerative process is a primary etiological factor in the large-lunged type of the disease is now considered highly improbable, though the theory is accepted as largely explaining the causation of the small-lunged type of emphysema which occurs in the aged. More weight appears to be attached to the theory promulgated by Cohnheim, that the disease is the result of an hereditary or congenital error in the development of the coiled elastic fibres entering into the composition of the terminal bronchi and surrounding the mouths of the air-cells and the walls of the infundibula.

Freund suggested that in some cases the primary etiological factor was an enlargement of the thoracic space from changes in the costal cartilages, which compelled the lung-substance to distend so as to fill up the increased space.

B. J. Collingwood has recently made a valuable contribution to the elucidation of the causes of emphysema. He rightly insists upon the importance of differentiating between the protected and unprotected portions of the lungs, and brings strong arguments to prove that in the unprotected

parts the emphysema must be the result of a violent *ex*piratory force, whilst in the protected regions the *in*spiratory force must be the factor at work. In coughing, the preliminary deep *in*spiratory effort causes hyper-distension of the infundibula in the protected parts; whilst later on, during the *ex*piratory effort, the protected infundibula, although containing air at a positive pressure, are contracted and contain less air than before, some of the air being driven into the unprotected parts of the lung.

The results of his experiments seem to demonstrate that a *general* emphysema must be chiefly *inspiratory* in origin, and his conclusions harmonise with, or effect a compromise between, the rival inspiratory theory of Laennec and the expiratory theory of Jenner and Mendelssohn; whilst they recognise the factors of weakness, either congenital or acquired, as a predisposing cause, they leave room for the recognition of *over-straining* of the elastic tissue as an important agent in the pathogeny of the affection, both in the protected and unprotected portions of the lung.

The *post-mortem* appearances of large-lunged emphysema are characteristic. The lungs do not collapse on opening the thorax, but may bulge out through the opening. The heart may be entirely covered by the thick margin of the left upper lobe, which lies in contact with that of the opposite side, and the diaphragm is considerably depressed. The lung is dry, soft, pale, and bloodless; it pits on pressure, is inelastic, and has a downy feel to the touch instead of yielding the crepitant sensation peculiar to the normal lung-tissue. Upon its surface, especially at the anterior margins, distended masses of lung-tissue, consisting of numerous coalesced vesicles, some of these as large as a filbert, may be seen, which collapse when cut into, and show irregular air-chambers or spaces with tags of fibrous bands, which were originally interlobular septa. There is usually some dilatation of the bronchi, but only occasionally bronchiectasis; atheroma of the pulmonary artery is commonly present, and in advanced cases dilatation or cardiac hypertrophy, with congestive swelling of the liver, and not rarely interstitial nephritis.

Both lungs are always involved, but one may be much more seriously altered than the other. The microscopic characters show the destruction of the air-cells and the disappearance of their walls and capillaries in the portions of the lungs affected. The elastic fibres show granular degeneration in those which remain; and imperfectly developed fibrous tissue at places marks the site of the normal interlacement of elastic-tissue fibres in air-cells not totally destroyed.

Symptoms.—The most obvious is dyspnœa, which at first is noticed only after exertion or during repeated attacks of catarrh; but in advanced cases it becomes persistent, though

liable to acute exacerbations, in which orthopnœa is usually marked. Such attacks when first seen are often mistaken for true asthmatic seizures. The breathing is wheezy and the dyspnœa is of the expiratory type, the expiration being prolonged and much laboured. Owing to the improper aëration of the blood, caused by the destruction of air-cells and obliteration of the capillaries, cyanosis may be extreme even whilst the patient is capable of moving about—a diagnostic point insisted upon by Osler.

Cough may only be a noticeable symptom in winter or during bronchial attacks, but ultimately it becomes as persistent as the cyanosis and dyspnœa.

In the emphysema which so constantly follows the bronchial irritation produced by breathing the dry dust of flax in the spinning-mills, the writer has been surprised on finding how seldom complaint is made of cough. The patient will often affirm that he never coughs, though during a brief examination he may cough several times: he has become so accustomed to his cough for years that he seems to be unconscious of it. This is of importance in the diagnosis. The presence and characteristics of the sputum depend upon the nature and extent of the accompanying bronchitis, which is seldom, if ever absent; sometimes hæmoptysis occurs.

True asthmatic seizures may from time to time supervene during the course of the affection; they may occur quite independently of any fresh catarrh, as when the patient changes from the locality in which he has been residing.

The skin is cool, and the temperature generally slightly subnormal, with a low-pressure small pulse. Dyspeptic symptoms and constipation, which are often present, greatly aggravate the dyspnœa, and ultimately the patient emaciates greatly; anasarca of the legs and albuminuria generally appear towards the termination of the case.

The *physical signs* in emphysema of the large-lunged variety are so characteristic that the condition can hardly be mistaken for any other. The physiognomy is easily recognised: the deeply furrowed, anxious, cyanotic countenance, with its prominent suffused eyes and thickened lips, the prominence of the scaleni and sterno-mastoids in the short thick neck, with the rounded and stooping shoulders, all go to form a picture which is quite unmistakable. Clubbing of the fingers, and even of the toes, is marked in all advanced cases.

On *inspection* the chest is seen to be barrel-shaped, the antero-posterior diameter being markedly increased; the sternum is thrust forwards, and the costal cartilages unduly prominent; the ribs are more closely approximated and more horizontally placed in the upper part of the thorax, whilst in the lower the intercostal spaces are widened and often depressed in inspiration. The shoulders are rounded and the

scapulæ horizontal. When at rest the thorax resembles that of a patient who has taken a moderate inspiration. During respiration the movements are obviously much restricted; even when the accessory muscles are in strong action the clavicles are prominent, and the hollows above them well marked, though these bulge outwards on coughing. The expiratory movement is lengthened and markedly laboured; during inspiration there is often marked recession of the lower portion of the thorax.

The veins in the neck are prominent, and may show pulsation; often an oblique belt of small distended veins is visible over the lower costal cartilages, marking the site of the attachments of the diaphragm. The nipple is raised, and the cardiac impulse is generally not to be seen in its normal position, but pulsation is visible at the ensiform cartilage or in the epigastrium.

On *palpation* the cardiac impulse is usually not to be felt owing to the emphysematous pad of lung in front of it, but a systolic heave or thud is evident over the ensiform. Vocal fremitus is lessened.

On *percussion* the note is hyper-resonant throughout, and sometimes approaches the tympanitic. Cardiac, hepatic, and splenic dulness are absent or much diminished, resonance being often found from above the clavicle to the costal arch in front, and from the supra-spinal fossa to the twelfth rib behind. The limit of cardiac or hepatic dulness, when present, is scarcely appreciably altered by forced inspiration or expiration. When the apex of the heart can be located by percussion, it is found displaced outwards and downwards.

Auscultation reveals a weak, short, inspiratory sound, which may be almost inaudible; whilst the expiratory sound is long, low-pitched, and wheezy, and may in the extent of its duration be three or four times that of inspiration. But more important even than the prolongation of the expiratory murmur is an obvious characteristic frequently overlooked—both murmurs have lost the peculiar features of normal breath-sound, the *vesicular* or *breezy quality having disappeared.* Râles and rhonchi are usually present, and may in some regions entirely replace the characteristic murmurs. Vocal resonance is variable. The heart-sounds, heard through the layer of emphysematous lung, are weak, and even when clearly audible they give the idea of being far off. If hypertrophy of the right ventricle be present, the first sound is strong and prolonged, but it becomes flapping and short in dilatation, and it may be accompanied by a tricuspid regurgitant murmur. The pulmonary second sound is usually accentuated. The heart being somewhat displaced downwards, the sounds are heard in their maximum intensity a rib's breadth lower than the normal position at the base.

Diagnosis.—There is little difficulty in recognising the disease, inspection alone often determining the condition, The cause or underlying affection should be determined by the history of the case, as chronic bronchitis and engagement in occupations involving strain.

The only difficulty, when any exists, will be found in eliminating cases of true *failing compensation* from arterio-sclerosis, or valvular disease, accompanied by marked dyspnœa. When there is no emphysema, the type of the dyspnœa should settle the problem, the feature in cardiac difficulty of breathing being its rapidity and paroxysmal character and the absence of the laboured expiratory efforts. The history alone will distinguish those cases of true pulmonary emphysema which are associated with cardiac dilatation.

Pneumothorax, when this does not arise from the rupture of a bleb or bunch of coalesced air-vesicles on the surface of an emphysematous lung, has been sometimes mistaken for the disease under consideration. The sudden onset, unilateral character, with immobility and bulging of the affected side, the rapid and extreme displacement of organs, the distinct tympanitic quality of the percussion-note, and the amphoric character of the auscultatory breathing, with metallic tinkling and succussion, are obvious distinguishing features.

Prognosis.—The disease is, as a rule, progressive and incurable ; but if the underlying cause—*i.e.* bronchitis, asthma, strain, &c.—can be remedied or removed, the condition may remain stationary for many years and give rise to no trouble save on exertion. In young subjects, where a rapid yielding to ordinary bronchial catarrh suggests the suspicion that there is either deficiency in the quality or quantity of the vital rubber in the terminal bronchioles and air-cells, the outlook is always grave. As a general rule, the prognosis is that of the bronchial inflammation causing and accompanying the emphysema ; the presence of bronchiectasis and fibroid phthisis deepens the prognosis. Where the heart is already involved, as evidenced by hypertrophy or dilatation, the case is grave ; and when venous pulsation, anasarca, and albuminuria occur, the end is sure not to be far off.

Treatment.—The clear indication is to treat the bronchitis, asthma, or other cause of the disease, and to insist upon the patient changing his occupation if this be one likely to produce increase in the intrathoracic air-pressure. Prevention of future bronchial attacks is of vital importance, and where the means of the patient will permit he should be directed to live permanently or winter in a warm climate. There is no drug which has any direct or specific effect upon the emphysematous lesion itself, but remedies which improve the tone of the muscular elements in the fine bronchioles, and which have an alterative action upon the bronchial epithelium,

will do good ; strychnine, cod-liver oil, and arsenic are unquestionably serviceable. The expectorant remedies suitable for bronchial inflammation and asthma will be found under 'Bronchitis.' There is, however, one drug which is of primary importance, as it is indicated in most of the dry catarrhs and in those where there is abundant adhesive secretion. This is iodide of potassium or sodium in full doses ; it is, moreover, the best anti-asthmatic we possess. Iron is often useful in very chronic cases. Constipation and indigestion, by increasing the abdominal tension, greatly aggravate the condition, and failing compensation embarrasses the breathing also ; these three conditions demand prompt and continuous attention. Cyanosis in association with paroxysms of severe dyspnœa, if not relieved by inhalations of oxygen, may be met with venesection or a rapidly acting strong saline purgative. The method of treating emphysema by causing the patient to breathe compressed air has given excellent results in the hands of Fowler and others at the Brompton Hospital. A cabinet or other form of compressed-air bath may be used ; the increased pressure need not amount to more than two-thirds of an atmosphere. The sitting should extend over at least two hours at a time, and the best results have been obtained by continuous residence for days in the air-chamber. The method of expiring into rarefied air after inhaling slightly compressed air is theoretically indicated, but it does not give the very decided beneficial results which are procurable by the use of the compressed-air chamber. The bronchitis and asthma are markedly relieved at the same time by the last-mentioned method of treatment.

Small-lunged emphysema, also called *atrophic emphysema*, or *senile atrophy of the lungs*, is part of the general wasting or withering of old age, though it may be more marked in the pulmonary tissue ; hence its name. Its consideration should not be separated from the large-lunged variety, as there is probably always the same cause of intrathoracic high air-pressure acting upon pulmonary tissue whose elastic and epithelial elements have wasted or atrophied.

The thorax is small, its walls are rigid, and the lower ribs almost in contact or overlapping each other, and very oblique, though the upper intercostal spaces are exaggerated. The lungs are small, very dark in colour, easily compressed, and contain little blood. The septa all through the lungs are wasted, so that many of the air-vesicles coalesce, forming large irregular air-chambers which may show withered air-cells about their margins. The bronchial walls show atrophic changes which often lead to some degree of dilatation.

The respiratory symptoms are not so marked as in the previously described variety, because less breathing-power and a smaller aërating surface meet the requirements of the

withered frame; hence, the respiratory movements are even more restricted, and the shrunken barrel-shaped chest shows a greater degree of fixation, owing to the ossification of the costal cartilages. The physical signs are of the usual emphysematous character; but, owing to the marked wasting of the pulmonary tissue, the area of cardiac and hepatic dulness may be increased instead of being diminished. This condition is uninfluenced by any form of treatment.

Acute vesicular emphysema is sometimes described as a separate variety of the disease; it occurs in severe asthmatic attacks, in angina pectoris, and often in acute bronchitis, and in epileptic and other convulsions. The absence of atrophic changes in the walls of the terminal bronchi permits of the over-distension passing away upon the subsidence of the temporary cause. The condition affords an explanation of the diminution or obliteration of the cardiac area in severe attacks of dyspnœa.

When the attacks, from whatever cause, are repeated, the permanent wasting of the septa with the final establishment of ordinary emphysema is the rule. The same result occurs in the next variety of the disease, the compensatory emphysema being at first of the temporary acute vesicular form, which gradually passes into the permanent variety.

Local or *compensatory emphysema* is usually found in the neighbourhood of chronic tubercular lesions, especially when these have undergone fibroid or cicatricial changes. This is seen most commonly at the apex of the lung, and is often associated with pleural thickening and adhesions. It may involve the anterior margins of the lungs after pleuritis has eventuated in extensive adhesions, and it is not uncommon in the scapular region, where a large cavity occupies a considerable portion of the apex in front. In extensive cirrhosis of the lung emphysematous tracts are constantly found scattered throughout the diseased areæ enveloping the cicatricial airless growths.

The importance of the condition lies in the fact that it may very materially alter the physical signs in pulmonary phthisis.

EMPYEMA

Inflammation of the pleural membrane eventuating in the formation of pus will be discussed under 'Pleuritis,' as the affection is but a phase or variety of pleural inflammation; only the distinguishing features need be dwelt upon here.

Etiology.—All ages from infancy upwards are affected, but the disease is more frequent amongst young males. In a small percentage of cases the presence of pus supervenes as a sequela of ordinary pleurisy with effusion, and in the merest

fraction of these cases it is induced by tapping with instruments which are septic.

In the majority of cases of pleuritis in children, and in a small percentage in adults, the effusion is purulent from the commencement. The exciting cause may be traumatic, as wounds, fractured ribs, &c., which introduce septic organisms from without; or the rupture of tubercular cavities in the lung, or gangrenous or cancerous growths, or subphrenic abscesses may infect the pleura.

The common cause, however, is the insidious secondary inflammation produced by the specific poisons which cause the infection of influenza, scarlatina, typhoid fever, pneumonia, pulmonary tuberculosis, and more rarely measles and pertussis.

The three bacteriological organisms which are commonly found in ordinary fibrino-serous pleurisies may be found in empyema. These are the pneumococcus, streptococcus, and tubercle bacillus. They may be found alone or in conjunction with each other; thus in tubercular pleurisy which becomes purulent streptococci only may be found, though probably the bacillus of tubercle is also present.

In adult empyema the organism usually present is a streptococcus, whilst in the empyema of childhood the pneumococcus is generally found. When neither of these is detected, which is seldom, the case is probably of tubercular origin.

Symptoms.—These are practically the same as those seen in acute pleuritis, but the chief point requiring emphasis is that there may be no evidence of onset, the disease, as is often the case with the secondary infections, being for a long time *latent*.

Shivering, pain in the side, fever, sweating, painful cough, and dyspnœa may be present early in those cases with rapid abrupt onset, but there are no differences in the constitutional symptoms, even in these acute cases, which will differentiate the disease from ordinary acute fibrino-serous pleurisy. The fever in empyema may be lower than in ordinary pleurisy, and even all through the attack it may be absent, especially in fistulous cases; but chills, sweating, diarrhœa, with an irregular or intermittent type of pyrexia, are suggestive of pus.

A very valuable diagnostic sign is the presence of a marked *leucocytosis*, and indican may be found in the urine.

The *signs* are those described under 'Pleuritis,' but some important modifications of these may be detected in empyema. Thus the bulging of the intercostal spaces, especially in children, is usually much more evident, and the enlargement of the affected side is generally more obvious. The veins may be conspicuously distended, and local œdema is common. At a later stage a local swelling, accompanied by redness,

tenderness, and fluctuation, may mark the advent of the approach of the pus to the surface of the body, constituting *empyema necessitatis*. The spontaneous rupture most commonly occurs in front in the fifth interspace, but there may be several such, which leave fistulous openings.

The displacement of liver and heart is more marked than in ordinary pleurisy with the same bulk of effusion. In rare cases there is marked pulsation (*pulsating empyema*) over the effusion, especially when this is large and on the left side; though it has been noticed in fibrino-serous pleurisy, the case is much more likely to be purulent if this sign develops during the course of the disease. It is not uncommon before spontaneous rupture through the skin; the pulsation is systolic, and usually seen in the vicinity of the heart, rarely posteriorly Baccelli's sign may be present—*i.e.* the *whispered* voice-sound is not transmitted through the purulent liquid; in *children*, when the effusion is even moderately large there may be loud bronchial or tubular breathing audible over it.

The course of the disease varies; as a rule the pus is not absorbed, but in children absorption may occur in some cases; once the writer demonstrated the absorption of a moderate empyema in an adult, but the possibility of such a result should never be counted upon. As already mentioned, spontaneous opening through the skin may occur, and recovery may follow without further interference, though convalescence may be prolonged for many months or even years.

The pus may rupture into the œsophagus, stomach, pericardium, or peritoneum, or it may pass down along the spine, and point as a psoas abscess in rare cases. Much more commonly, however, it finds its way into the air-passages, either by slow ulceration of the pulmonary tissue and burrowing a considerable distance under the visceral pleura, or more directly by perforating a bronchus, in which latter case pneumothorax (*pyo-pneumothorax*) is likely to occur. Many cases undoubtedly recover spontaneously in this way when air does not enter the pleural cavity.

Morbid Anatomy.—This in the main corresponds with the changes seen in pleurisy, but in addition to the accumulation of fluid being always purulent, or sero-purulent, it may in some instances be found offensive and sometimes indescribably putrid. The pleura is always much thickened and covered with a greenish-grey or white layer of lymph; excoriations, gangrenous patches, or perforations are often visible. The lung is usually much compressed or carnified. Loculated purulent accumulations may be found; when free air has previously entered the pleural cavity it may rush out through the pleural incision. The displacement of the mediastinum and pericardium is very obvious.

Diagnosis.—All the points in the differentiation of purulent from ordinary fibrino-serous pleurisy have been already mentioned, save the one which should never be omitted—*i.e.* exploratory puncture. This can usually be accomplished by the insertion of the ordinary hypodermic needle, a few drops of sterilised water being first injected to prevent clogging of the needle by blood or shreds of lymph.

Prognosis.—This will depend upon the nature of the primary condition: when the pneumococcus is present in the pus the look-out is more favourable than when streptococci are found; sterile pus generally means tuberculosis. Children as a rule recover rapidly, but nothing influences the prognosis so favourably as early recognition and prompt surgical interference, though recovery, as already mentioned, often follows spontaneous evacuation by the skin or bronchial route; this should never be permitted to occur. All authorities agree in emphasising the fact that, no matter how hopeless the case may appear when first seen, the patient should get the chance of an operation, as there is always some prospect of recovery after thorough drainage has been once established.

Treatment.—There is no excuse for delay, as soon as the presence of pus is demonstrated by the needle. If the microscopic examination reveals the presence of the pneumococcus in a child, with absence of any grave constitutional signs of sepsis, aspiration of the pus should be immediately resorted to, there being a fair hope that recovery may follow the first or second tapping. In all other cases a free incision should be made without delay in the sixth or seventh interspace in front of the posterior axillary line, or in the eighth or ninth in the line of the angle of the scapula. By this free incision the empyema is treated like a large abscess, and the most thorough drainage secured by the introduction of the largest possible rubber tube, with a flange upon its extremity to prevent its slipping into the pleural cavity. Where the chest-wall refuses to fall in, or where the lung fails to expand, excision of portions of several ribs by Estlander's operation, or the more radical procedure of Delorme, may have to be resorted to. Irrigation, even in foul cases, is seldom necessary. (For the different methods of securing drainage by continuous syphonage, and for details of the general management of empyema cases, see the author's 'Dictionary of Treatment,' pp. 266–270.) Expansion of the compressed lung may be assisted during the healing process by deep inspiration-exercises steadily persevered in several times daily.

ENCEPHALITIS.

(See CEREBRAL INFLAMMATION, p. 186.)

ENDOCARDITIS

Inflammation of the lining membrane of the heart is an infective disease, generally of secondary origin. In the great majority of cases the inflammatory action is limited to that portion of the endocardium which covers the segments of the valves; practically, therefore, it may be considered as a *valvulitis*. The disease appears in three forms, which often can be clinically recognised. These are—(1) *Acute, simple, rheumatic*, or *benign* endocarditis; (2) *Malignant, ulcerative*, or *infective* endocarditis; (3) *Chronic* or *sclerotic* endocarditis.

It has long been suspected that these different varieties are phases of a common pathological process, and recent bacteriological researches have practically established this view, which is in complete accord with clinical experience, since it has long been recognised that one variety—the simple—may in some cases pass into the malignant form. The simple form is very commonly witnessed as passing into the chronic, and it is almost certain that when life is sufficiently prolonged the malignant may also pass into the chronic form; whilst it is very common to observe the passage of the chronic variety into both the simple acute and malignant forms.

Etiology.—*Acute simple endocarditis* is in the great majority of instances associated with acute rheumatism. It is also found to accompany scarlatina, tonsillitis, chorea, typhoid fever, pneumonia, gout, Bright's disease, more rarely diphtheria, small-pox, erysipelas, phthisis, cancer, measles, and chicken-pox. The condition is very commonly seen to supervene upon the chronic form, which may have originally been associated with any of the above diseases, or which may have been the result of simple arterial strain. It is more likely to complicate acute rheumatism in children than in adults, and bears little relation to the severity of the attack.

Morbid Anatomy and Pathology.—The left side of the heart is far more commonly affected than the right, and the mitral valve suffers more frequently than the aortic. The lesion first shows itself near the margins of the valve, which are in contact when the opening is in the closed state, hence the extreme margins generally escape. Here minute warty or cauliflower vegetations appear, which rarely exceed from $\frac{1}{20}$ to $\frac{1}{6}$ in. in diameter; they are often pedunculated, and by their minute dimensions contrast with the larger masses found in malignant endocarditis, though there may be some slight sub-endothelial swelling, necrosis, and cell-infiltration. These latter changes are, however, when present, probably evidence that the simple is passing into the malignant form, because in the typical simple valvulitis of acute rheumatism there is practically no visible redness, swelling, or necrosis,

and the other parts of the endocardium, with the exception of that covering the chordæ tendineæ, are entirely free. The vegetations at first consist of blood-elements—*i.e.* platelets and white cells with fibrin and micro-organisms, which may be swept onwards into the vessels ; ultimately they become converted into connective or granulation tissue, and are generally covered with a layer of fibrin, which may at times detach itself and be swept into the circulation as an embolus. These vegetations may entirely disappear, but they frequently leave behind them such changes in the delicate structures in the valve as may lead to shortening, puckering, or deformities, which eventually may end in permanent obstructive or regurgitant valvular disease.

The bacteriology of the vegetations has of late years received much investigation, a frequently present organism being the *Diplococcus rheumaticus*, which is believed to be the cause of acute rheumatism ; but streptococci, staphylococci, pneumococci, and the microbes of typhoid fever, tubercle, gonorrhœa, and influenza have been often demonstrated.

Symptoms.—These, as a rule, are latent, and there may be total absence of evidences of any cardiac complication, the endocarditis being only discovered on *post-mortem* examination. The advent of simple endocarditis may, however, in cases of acute rheumatism, be suspected when the heart's action gets weak and irregular without obvious cause, and where palpitation is felt. The physical signs are also fallacious ; there is usually no marked increase in the pyrexia, but the presence of a harsh rough first sound, in a case which a few days previously had exhibited a pure systolic sound at the apex, should excite strong suspicion, especially should this deepen into a distinct murmur. In the experience of the writer a marked alteration in the cardiac rhythm is a more reliable sign than the presence of a systolic murmur, which may depend upon blood-conditions. The diagnosis is tolerably certain if both signs exist together, even should the rhythm soon become normal again. The mitral-area murmur is more suggestive than a bruit heard over the aorta, and a pulmonary murmur may be discarded. The murmur is soft and its area circumscribed.

In acute rheumatism, where the valves have been permanently damaged in a previous attack, the advent of a fresh endocarditis is usually beyond the reach of diagnosis unless the condition should pass into the malignant type. In a considerable number of cases, especially in children, pericarditis supervenes upon the endocarditis, and this must be differentiated from a local pleuritis.

Prognosis.—This is favourable as regards the immediate outlook, since the valvulitis itself does not tend to the production of a fatal issue, which is only likely to occur in case

pericarditis or myocarditis supervene. The ultimate prognosis, is, however, another question. In a large number of cases the murmur entirely disappears, but in a considerable percentage of cases it remains, or permanent valvular disease ultimately follows. There is no means of forecasting which result will be most likely to occur, save one, which should always be explained to the patient's friends : ultimate freedom from valvular defect is much more likely to follow if the patient can be induced to take *prolonged* rest in the horizontal position after convalescence has been complete.

Treatment.—This in the first instance will be the treatment of the primary disease to which the simple endocarditis is secondary ; rest in bed and milk-diet will therefore be continued.

As a rule it is considered wise in acute rheumatism to suspend the salicylates as soon as the diagnosis of valvulitis is decided upon. The best drug-treatment will consist in the free administration of alkalies, as bicarbonate of potassium 1 dr. in solution added to fresh lemon-juice. Blistering over the cardiac area has long enjoyed the reputation of being both prophylactic and curative. Rarely are poulticing, ice, or morphia required, as cardiac pain is usually absent. At a later stage iodides in full doses seem to possess the power of aiding resolution. All measures are, however, of little worth compared with *rest* ; the value of this agent as a preventive of chronic valvular deformity has been referred to under Prognosis.

ENDOCARDITIS, CHRONIC

This exists in two well-marked forms. One is the sequel of the acute rheumatic endocarditis just described, which leads to deformities—thickening or coalescence of segments, producing stenosis and shortening or contractions of the valve-segments, which permit of regurgitation. The other form is the sclerotic or fibroid, produced by the causes which operate in inducing arterio-sclerosis, or atheroma, as prolonged muscular strain, syphilis, and poisons like alcohol, gout, over-feeding, &c.

Under ' Valvular Disease ' the results of chronic endocarditis will be dealt with, and need not be here detailed.

ENDOCARDITIS, MALIGNANT

This is also known as *Ulcerative* or *Infective Endocarditis*.

Etiology.—This disease, like simple endocarditis, is usually *secondary* to some other affection, as pneumonia, scarlatina, erysipelas, septicæmia, acute rheumatism, gonorrhœa, and

diphtheria. It occurs more frequently in association with pneumonia than with any other disease, and next in frequency perhaps with acute rheumatism. Previous simple endocarditis, which has eventuated in chronic valvular deformity, is an important determining factor in most cases. It is certain that a simple benign endocarditis may pass into the malignant type.

It seems equally certain that in a small percentage of cases the disease may be *primary*. The writer has seen it in its most typical form begin in an acute onset with rigors in an apparently perfectly healthy subject, and run its fatal course, without a symptom or sign which could be attributed to any other affection.

Its association with septicæmia is very much closer than ordinary statistics would seem to prove, since the condition of sepsis is excluded often because of the absence of wounds, the puerperal state, and other obvious causes of sepsis. The writer's experience satisfies him that the unhealthy condition of a mouth containing a number of foul suppurating cavities belonging to the molar teeth is a powerful determining agent in a considerable number of cases, and he believes that this is the probable factor in most of the simple cases which pass into the malignant form. Ewart reported a case several years ago, 'apparently due to infection from dental caries,' but stomatitis was also present in this case.

Its association with rheumatism will be referred to later on in discussing the pathology of the disease.

Morbid Anatomy and Pathology.—The changes resemble those found in the simple form, but the vegetations are usually larger, and assume an exuberance or massive growth which is almost characteristic. There are always evidences of necrosis or loss of substance in the valve and surrounding endocardium.

The researches of Poynton and Paine have demonstrated the presence of the *Diplococcus rheumaticus* in several instances, thus proving that malignant endocarditis in at least a percentage of its cases is caused by the same microbe which produces acute rheumatism and its apparently benign endocarditis. The cultivation and subsequent experimental methods adopted proved this fact beyond doubt, and these observers state that the only essential difference between the two processes is that whilst in the simple or benign the diplococci disappear entirely from the diseased tissue as soon as necrotic changes appear, in the malignant form they are found in masses buried in the necrotic tissue.

The causes which increase the virulence of the diplococcus are unknown. Singer maintains that this diplococcus is but an attenuated form of streptococcus pyogenes or other pyogenic cocci.

In addition to the *D. rheumaticus*, many other pus-forming cocci are met with in malignant endocarditis, notably the pneumococcus, staphylococcus, streptococcus pyogenes, micrococcus lanceolatus, and all the forms found in the simple or benign type, including the bacillus coli and the influenza and gonorrhœa organisms.

In addition to the ulcerative lesion in the valves and in the neighbouring endocardium, patches of disease are constantly found on the septum between the ventricular cavities and on the wall of the left auricle. The valve may be perforated, a portion of it may become entirely detached, or a valvular aneurism may be formed and the cordæ tendineæ are sometimes necrosed; the disease may extend into the coats of the aorta and pulmonary arteries, especially when a pedunculated fragment of a necrosed valve is continually flapped against the arterial walls by the blood-stream during systole. Though the disease is much more commonly limited to the left side, not infrequently the right heart is also involved, and in a small percentage of cases it is found confined to the right auricle and ventricle.

As already mentioned, malignant endocarditis is frequently found associated with long-standing sclerosed valvular lesions. Death is usually caused by embolism; the vegetations, consisting of the micro-organisms with blood-elements, and covered with thick layers of fibrin, are swept into the *arterial* system, causing plugging of the vessels in distant parts, as described under 'Embolism' and 'Thrombosis,' and eventuating in gangrene of the extremities, infarcts in the kidneys and spleen, hemiplegia, cerebral softening or abscesses, intestinal thrombosis or embolism, and retinal blocking.

Multiple abscesses may be found all over the body, as in pyæmia, and suppurative inflammation of the serous membranes—*i.e.* pericarditis, meningitis, and empyema—may be produced. When the right side of the heart is affected there may be pulmonary infarcts and multiple abscesses. Suppuration of the parotid and hepatic abscesses are not infrequent.

Symptoms.—These are very varied, and in some instances may be long latent; the characters of the primary affection to which the endocarditis is secondary often mask both the symptoms and signs of the valvulitis. Usually, however, the primary fever becomes intensified and altered in its type; it may get markedly irregular, and chills or severe rigors followed by profuse sweating, some delirium or stupor, dyspnœa, progressive muscular weakness, and emaciation follow.

The various complications caused by the lodgment of the septic emboli in distant organs and in large arteries constitute the most characteristic part of the clinical picture, though it must be remembered that malignant endocarditis may

occasionally run its course without the phenomenon of embolism being apparent. Septic rashes often appear, and sometimes jaundice is present; always marked leucocytosis can be demonstrated. All the above symptoms may be present in primary cases, and occasionally the type of the fever may be continuous.

Cardiac signs may be absent in some cases, and in those occurring in subjects possessing old valvular lesions the signs of the valve-destruction are usually wanting, or may be misleading. The presence of recent murmurs at the mitral or aortic area are highly suggestive when considered with the gravity of the constitutional symptoms. In the writer's experience the presence of an *aortic regurgitant* murmur, appearing under observation in a patient suffering from an irregular, remittent, or intermittent type of fever, is a diagnostic point of such value and importance that he has been able to make a diagnosis in the absence of all other signs and symptoms save the presence of pyrexia. It is of equal significance when it suddenly appears in an old case of mitral disease associated with grave signs of severe septic fever. Tricuspid murmurs are also of great value in diagnosis.

The duration of the disease is as variable as its symptomatology. It may be fatal in three or four days, or it may last for many months ; the majority of cases terminate fatally in about five or six weeks from the onset.

Several types of the affection are met with, and four are usually described, though one type may run into another, and occasionally a case may at different stages of its progress show the chief clinical features of each type.

Cardiac Type.—As its name implies, the cardiac symptoms and signs predominate. Such cases nearly always occur in subjects possessing old valvular deformities ; and though the malignant affection may begin suddenly with abrupt invasion, it may drag out a course of many months. (These latter cases, however, had better be considered as examples of recurring attacks.) The fever is of an irregular or remittent character, and chills are common, followed by more or less sweating—but this may be profuse, especially at night, without any chills. The connection of this type with rheumatism is close, as not only is there the history of former rheumatic attacks, but not infrequently rheumatic symptoms may suddenly walk across the stage during the exhibition of the malignant phenomena, though they always play a minor part in the progress of the case. Some of the examples of this type certainly appear to begin in the benign form, and to deepen into the malignant. When such end in recovery they are generally classed as having remained benign all through. This is a mistake, as Ewart has found after death almost complete recovery of the parts affected in the earlier stages of

the attack, and the writer once witnessed a similar striking result.

The original murmurs may change somewhat in character, but the appearance of a fresh aortic regurgitant bruit is of great moment in diagnosis. Hence the value of a daily stethoscopic examination. Cardiac distress and dyspnœa generally appear soon. Once the writer was fortunate in witnessing the abrupt appearance of a mitral regurgitant murmur which replaced a long-standing typical murmur of mitral stenosis. He had frequently, some years before, exhibited the patient at his clinic, and after the fatal termination of the attack of malignant endocarditis, during which the alteration took place, he was still more fortunate in procuring the specimen which was proved by Lorrain Smith to be an example of ulcerative endocarditis, ending in necrosis of a portion of the narrow funnel-shaped valvular opening, which converted the case from one of stenosis into one of typical regurgitation. The symptoms and signs of mitral regurgitation had been observed for a period of nearly three months before death. There was also very distinct evidence of an effort at repair in the neighbourhood of the necrosed or ulcerated spot.

At any period of the progress of the disease in the cardiac type, rigors and other septic manifestations may show themselves, embolism may supervene, and the disease may run a swift course. In lingering cases the emaciation becomes so extreme that the disease may be mistaken for phthisis.

Typhoid Type.—This is the most common form, and, as implied in the name, the fever is of a more or less continuous character. There are rapid prostration, diarrhœa, abdominal tenderness, petechial eruption, somnolence, delirium, sweating, and all the usual features seen in enteric fever, for which it is often mistaken till the advent of arterial plugging in some distant part reveals the true nature of the disease. Usually after this occurs, a remittent or irregular temperature becomes evident; some dyspnœa and parotitis may be present early. There may be at first no cardiac sign or symptom to arouse suspicion, save the evidence of old valvular lesions, and their subsequent compensatory changes. The pulse is, however, always rapid, and in the majority of cases cardiac pain and distress with dyspnœa soon make their appearance.

Pyæmic or Septic Type.—This is met with under the usual pyæmic or septic conditions, as in puerperal infection or septicæmia in association with suppurating wounds, empyema, gonorrhœa, pneumonia, and scarlatina, &c., rarely in association with acute rheumatism, but sometimes in patients with old valve-lesions, and sometimes it appears as a primary disease.

The form of the fever is distinctly from the first intermittent. The onset is ushered in with a severe rigor, followed by hot skin, and afterwards by copious and exhausting perspiration; the rigors recur, and during their existence a hyperpyrexial temperature may be registered. This type was formerly described as an arterial pyæmia, the only signs being rigors and plugging of the arterial branches throughout the body. The infective emboli lead to the formation of infarcts, gangrene, true pyæmic abscesses, brain-softening, and all the symptoms described in the article on 'Embolism' on page 356. The renal, hepatic, intestinal, splenic, and other symptoms need not be here again described. They are common to all the types of malignant endocarditis, but are met with in their most characteristically sudden forms in the pyæmic variety. The valvulitis may obviously be one of the late manifestations of a true pyæmia in rare cases. A form of malignant endocarditis has been described under the title of a *malarial type*. This is simply the true septic or pyæmic variety when met with in its most typical form. It has no connection with malaria, but the symptoms may be identical till embolism occurs, and there may be no cardiac signs whatever. The writer lately witnessed such a case with Drs. Rowan and Hillyard, in which a profound rigor occurred at exactly the same hour every evening for many days in an otherwise healthy patient, the temperature always slightly exceeding 106° F.; in a few hours after each rigor the patient invariably expressed himself as feeling quite well, and continued so till the following evening, with a typically normal pulse and temperature. There was a hesitation at first in excluding malaria, as the patient had been in a malarious zone within two years before; but the sudden appearance of an aortic regurgitant murmur, caused evidently by necrosis of a semilunar valve, confirmed the diagnosis of malignant endocarditis; extensive arterial plugging afterwards occurred. There was no apparent focus of infection in this case, which may be taken as a typical example of the difficulty in diagnosis. Dreschfeld uses the term 'malarial' as a synonym for the cardiac type; but the cardiac cases with malarial symptoms which he describes would be better called cases of *recurring* or of *chronic vegetative endocarditis*.

Cerebral Type.—This rare form may begin with abrupt onset in which violent headache, delirium, meningeal and spinal symptoms, with hyperpyrexia, are prominent. The cases run a very rapid course, ending in coma. The clinical picture may be identical with basilar tubercular meningitis, or with cerebro-spinal fever. Such cases sometimes supervene upon acute pneumonia, or arise during convalescence from this disease.

Diagnosis.—The points of importance have been already emphasised in the above brief description. As a rule, there

should be no difficulty after embolism has occurred. In the early typhoid cases, the sudden onset, some breaks in the continuity of the pyrexia, the supervention of rigors, cardiac distress and dyspnœa, and the absence of the Widal's reaction are differential points which with the presence of an old valvular affection, or a recent murmur, are fairly conclusive; but acute tuberculosis is not so easily excluded.

The greatest difficulty will be found in excluding severe benign endocarditis in rheumatic cases at an early stage; this amounts to an impossibility in those cases where the benign is passing into the malignant form. As already stated, the essential difference, as demonstrated by Poynton, lies in the fact that in the benign form the valve-tissues are able to destroy the diplococcus rheumaticus, whilst the resistance of the living cells fails in the malignant form. The *rapid development of a profound anæmia with emaciation* may indicate that cell-resistance has become exhausted. If so, the condition of the blood should be carefully watched. Leucocytosis is always present in the malignant form, and the polynuclear cells are always increased.

The so-called 'malarial' type affords one of the most interesting and difficult problems in diagnosis. Malaria is, however, easily excluded by a blood-examination, which will not reveal the parasites of malaria (there is always leucopenia present in malaria); the value of the sudden appearance of an aortic regurgitant murmur has been already mentioned, and the importance of an examination for a focus of infection in any part of the body, even in necrosing teeth, is obvious.

The following list of diseased conditions in which a paroxysmal or remitting pyrexia is to be met with is a valuable contribution to diagnosis made by the late Dr. Ord. The diseases are given in the order originally enumerated by him —viz.:

(1) Ague; (2) remittent fever, malarial and non-malarial; (3) enteric fever; (4) relapsing fever; (5) tuberculosis; (6) confined pus; (7) pyæmia; (8) septicæmia; (9) *ulcerative endocarditis*; (10) lymphadenoma; (11) leucocythæmia, occasionally; (12) irritation of the biliary passages by impacted gall-stones or other causes; (13) irritation of the urinary passages; (14) irritation of the alimentary canal; (15) syphilis; (16) morphia-habit; (17) foreign bodies impacted in the tissues. The greatest difficulty, he maintained, was to be met with in distinguishing between ulcerative endocarditis and tuberculosis.

The presence of streptococci in the blood is a valuable point in diagnosis and treatment, but the diplococcus rheumaticus is not to be found usually in the blood, being confined to the tissues.

Sicard, in the investigation of the bacteriology of the disease, has recently found that in thirty-two cases the blood-

cultures failed to show any organisms in twenty-two cases; of the remainder four were due to staphylococcus pyogenes aureus, three to the pneumococcus, two to streptococci, and one was of mixed origin.

Prognosis.—The title 'malignant' implies a fatal issue, but that this is not correct there is a gradually increasing accumulation of clinical material to prove. The prognosis is, however, always grave; the slender hope surrounds the old rheumatic cases of the cardiac type. Sansom, Ewart, and Morley maintain that recovery does sometimes occur. The three other types may be considered as practically always fatal, though the malarial sub-type may be prolonged for many months.

Treatment.—The primary disease, to which the endocarditis is secondary in most instances, will call for prompt treatment, and the removal of foci of infection should be carried out, when possible, without delay.

Antiseptics are indicated, and Sansom has long maintained that the best remedy is sulphocarbolate of sodium in 30-gr. doses, accompanied by inunctions of carbolic oil (1 in 5). Ewart recommends protargol ointment, and suggests the intravenous injection of perchloride of mercury or colloidal silver. Quinine, benzoates, and other antiseptics are powerless. Chief interest centres round the use of injections of antistreptococcus serum; for ordinary use the best serum is a polyvalent one obtained by injecting the horse with cultures obtained from different sources. This is unquestionably the most rational treatment in those cases, especially of primary origin, where the streptococcus can be discovered in the blood; but such discovery is difficult and requires time, and for the serum to be of any use it must be employed early and frequently. Ogle, Spencer, and others advocate its use as soon as the symptoms warrant a diagnosis, and undoubtedly some cases appear to have been saved by it. It seems equally certain that it is useless in the infection of virulent rheumatic diplococci. It must be remembered that the serum is not antitoxic but simply bactericidal, and that it is but a chance that the animal which supplied it was dosed with cultures of the same streptococcus which is causing the disease demanding relief. Wright condemns the use of all the sera at present obtainable, owing to the danger of their diminishing or destroying the natural immunising effect of the body, recovery being rendered impossible when the phagocytic power is destroyed. Several undoubted cases of malignant endocarditis have been successfully treated by Wright's vaccine method.

ENTERITIS

As its name implies, this is an inflammation of the intestinal surface, generally regarded as of catarrhal nature. Various names are given to its varieties according to the portion of the tube affected, as jejunitis, typhlitis, colitis, entero-colitis, &c.

Specific forms of inflammatory action in the alimentary canal eventuate in diseases which are described under their distinctive names, as dysentery, typhoid fever, cholera, appendicitis, &c.

The group of cases usually described under the name of 'catarrhal enteritis,' practically corresponds with the different forms of diarrhœa, so that catarrhal enteritis and diarrhœa are generally regarded as synonymous terms. This loose nomenclature owes its origin to the fact that in the great majority of cases the only symptom of the enteritis is to be found in the presence of frequent liquid motions, whilst it is too often forgotten that both the diarrhœa and the enteritis (which latter, indeed, is often absent) are simply secondary results of the action of a chemical purgative which is a product of microbic or ferment activity.

The only justification for retaining the title 'enteritis' as covering the symptom known as diarrhœa lies in the probability that after the expulsion of the toxic purgative material a catarrhal condition of the mucous membrane may be left behind which probably might prolong the diarrhœic symptom. Under 'Cholera Nostras,' 'Colitis,' and 'Diarrhœa' will be found a brief description of the various conditions supposed to be the result or manifestation of a catarrhal inflammation involving the different portions of the intestinal mucous membrane. Cholera infantum and the so-called enteritis of infancy are referred to under the heading 'Diarrhœa in Children,' upon page 289.

ENTEROPTOSIS

This is a condition of general ptosis, or downward displacement or dropping of the abdominal viscera. It was first fully described in 1885 by Glénard, and is often wrongly called Glénard's *Disease*. It is also known as splanchnoptosis, and visceroptosis.

Etiology. — The abnormality is caused by a looseness or stretching of the ligaments or peritoneal folds which should hold the viscera in their natural positions. With this is often associated relaxation of the abdominal wall; hence the condition is common in women who have borne numerous children. It may, however, occur in young, thin, and single

women, in which case it is usually associated with neurasthenic symptoms of a distressing type. That the condition may exist to a marked degree without giving rise to any symptom for many years is generally recognised. Tight-lacing, the presence of peritoneal adhesions and hernia, violent exertion or straining, and rapid emaciation, following acute illness, have been regarded as causes.

Signs.—The displacements are, as a rule, easily made out in thin subjects by palpating the solid organs, which, owing to their weight, drop downwards when the body is in the erect position, and the recumbent posture brings them back to their normal places. In this way the margin of the liver may be felt below the umbilicus, and the spleen may be detected rolling or wandering about in any portion of the middle or lower zones of the abdomen; both kidneys may be often easily pushed out of their positions in the loins, but especially the right one. The stomach-displacement may be detected by inspection, and when the liver is low the smaller curvature of the stomach may be visible below the xiphoid, and its larger margin much lower than the umbilicus. Sometimes it assumes a vertical position towards the left side, and it may be markedly dilated from kinking or by mechanical obstruction of the fixed descending portion of the duodenum by the forward displacement of the kidney. In these latter conditions palpation, percussion, succussion, and inflation will demonstrate its situation when inspection fails. Intestinal displacement is often marked, the mesentery permitting of ptosis of the entire bulk of the small intestine, producing a characteristic pear-shaped deformity of the abdomen, which is narrow above the navel, and dilated in the lower zone. The transverse colon is often greatly displaced, being bent into a U, with the middle of the concavity as low as the pelvis.

The displacements or dropping of the different organs are often referred to under the following terms: hepatoptosis, nephroptosis, splenoptosis, gastroptosis, enteroptosis, and coloptosis. These are sometimes described as separate conditions, but unless when they are the result of adhesions, or displacements by tumours they may usually be regarded not as separate entities, but as part of the general pathological condition known as Glénard's disease. This is probably also true of displaced, floating, or movable kidney, which has the most obvious pretensions to be regarded as a special pathological condition, and which will be described separately under 'Kidney Displacements.'

Symptoms.—As already mentioned, these may be entirely absent, and not unusually they only commence after the patient has accidentally discovered or been informed of the abnormality. They consist of various sensations of depression or exhaustion, both mental and physical, from the

milder grades of invalidism to the most pronounced degree of neurasthenia. In some cases, however, the psychical element is entirely absent, and then a study of the mechanical effects of the displacement can be made without difficulty. Gastric dilatation may produce the typical symptoms of pyloric obstruction, but more usually vomiting, hyperacidity, pyrosis, and gastralgic pain are absent. Constipation may be as obstinate as in abdominal obstruction, and may not yield till the patient is compelled to take to her bed. Occasionally it is followed by diarrhœa, and often the motions are preceded by severe colicky pain. The unmistakable signs of kinking or torsion of the renal vessels and ureter, ending in temporary hydronephrosis or strangulation of the kidney, may be present. The dragging, wearying pains are usually aggravated by exercise of all kinds, and are relieved by resting in the horizontal position, so that a small percentage of the patients take to bed and become permanently bed-ridden. Generally marked relief may be obtained by the patient placing both hands with the fingers interlaced just above the pelvis, and *lifting* upwards and backwards the relaxed abdominal walls. It is not correct to say that pressure gives relief, as most authorities state; the manœuvre which gives relief is really one which removes the pressure.

Treatment. — In recent cases following acute emaciation and prolonged physical strain, as in the nursing of a relative through a fatal illness, it is possible that the condition may be removed by promptly sending the patient to bed and insisting upon prolonged rest and over-feeding, so as to restore the paddings of fat in the abdomen. In all other cases the only available treatment, besides dealing with the gastric, intestinal, and neurasthenic symptoms, is the adjustment of a well-fitting abdominal binder. Endless are the patterns of these; but the nearer a binder can be got to afford the kind of support which the patient instinctively applies to the lower portion of the abdomen with her own hands, when wearied by the dragging pain, the better will the result prove.

In cases where the suffering becomes intolerable, abdominal section and stitching of the displaced organs in their proper position should be resorted to.

In a severe case of the writer's, suppuration of a gall-bladder containing calculi precipitated a laparotomy; after excision of the gall-bladder the liver and kidney were fastened by Dr. J. Campbell with complete and permanent relief. Gall-stones are often the direct result of the hepatoptosis, and may call for active surgical treatment.

EPILEPSY

Though there are few diseases whose clinical manifestations are more sharply defined, Sir W. Gowers, in his masterly and philosophic article in Allbutt's 'System,' states that the attacks which constitute the malady present variations in degree and character so great as to baffle every attempt to frame a clinical definition of the disease.

The essential point in any definition of the affection must be the sudden loss or impairment of consciousness, with or without convulsions, and that this must be the result of causes which are independent of organic lesions of the cerebral matter, its vessels or membranes.

Pathology. — Epilepsy must be regarded in the present state of our knowledge as a neurosis or purely functional disease, and consequently it has no morbid anatomy, though Lewis, Chaslin, and others have recently reported marked changes in the cortical cells; these are probably the results and not the cause of the attacks. Of its pathology nothing definite and certain is known. It is usually held to be the result of discharges of nerve-energy caused by some unstable condition of the spongy grey matter of the cerebral cortex. Whatever may be the nature of the molecular action which precedes the discharge, whether it be chemical or electrical, it is useless to speculate; but as regards the site of the hyperexcitability or unstable equilibrium, clinical experience and the results of experiment render it almost certain that this is situated in motor centres in the cortex in those cases where convulsive muscular movements occur. Where only consciousness is lost or impaired, it is highly probable that the discharge takes place in the sensory centres, proceeding in an upward direction, so that their function is abolished. Ziehen maintains that the clonic spasms arise in the cortex, and the tonic in the sub-cortical centres; whilst others believe that the irritation which produces the discharge arises in the cortex, but that the discharge occurs from the sub-cortex. Irritation of the motor area of the cortex will produce the convulsions, but excision of a particular area will abolish the movements in the muscular groups connected with it. Turner maintains that the primary etiological factor in epilepsy is the presence of imperfectly developed and probably unstable nerve-cells, and that the immediate cause of the fit is an altered condition of the blood which produces clotting in the small cerebral vessels.

The theory that the epileptic attack is the direct outcome of some organic poison manufactured within the body is gaining ground, and receives some support from the results of experiments by Voisin, Krainski, and others in which

the urine of epileptic patients, when injected into the blood of animals, was found to be more toxic after an attack than before.

Etiology.—*Heredity.*—A family history of epilepsy is found in about one case in every three met with in private practice; and a history of either epilepsy or insanity is to be found in one out of every two cases. If other neuroses are included, a history will be obtainable in two out of every three cases. An hereditary history is more frequently found in females than in males.

Age.—Half the cases begin between the ages of ten and twenty years, one-eighth before the age of three years, and about three-fourths before the termination of the twenty-first year. The liability to epilepsy diminishes markedly after middle-life, and is very rare after sixty.

Sex.—The sexes are about equally affected when all ages are included, but after thirty the incidence markedly increases amongst males.

Infantile convulsions, usually associated with delayed dentition or rickets, may lay the foundation of a true epilepsy, which may not, however, appear for some years later, though in a small number of cases the convulsions recur at short intervals, the one condition passing directly into the other.

Alcohol.—Two points are probably very important as regards alcohol. The disease is common in the offspring of drunken parents, and drunkards themselves often become epileptic late in life. Both facts may be explicable upon the hypothesis of an inherited neurotic condition. Epilepsy probably occurs more frequently amongst absinthe-drinkers than amongst whisky-drunkards.

Traumatism seems to be the exciting cause in some cases, exclusive of all the Jacksonian ones; and there are some grounds for believing that injury to the cortex in difficult labour may lay the foundation in infants who do not show the other symptoms or signs of birth-palsy. Diffuse blows on the head may be the exciting cause in drunkards who exhibit no other sign of cortical injury.

Mental emotion seems to be the exciting cause of a fair percentage of cases, acting somewhat as it does in the production of hystero-epilepsy and chorea; terror and acute grief are the most potent emotional causes.

Infectious fevers—notably scarlatina—may be the cause of the first attack.

Reflex causes are not uncommon, as injury to a sensory nerve, worms in the intestine, cicatrices, &c. The writer saw one unmistakable case arise as the result of a mass of glazier's putty which filled up the auditory passage and became as hard as porcelain without producing inflammatory changes.

Syphilis, *gout*, and *phthisis* are often accredited as causative factors with but little evidence to support the statement, and masturbation and sexual excesses are more likely to be results than causes.

Symptoms. — Two types of the disease are met with—the severe, always associated with general convulsions, and known as *major* epilepsy or *grand mal*; and the *minor* form, called *petit mal*.

GRAND MAL

The patient becomes unconscious, falls down in a general tonic spasm, and a series of clonic convulsions affecting all the muscles of the body follow. Premonitory symptoms may be present for a day or two before the seizure. Such are dizziness, vertigo, muscular jerkiness, mental restlessness, irritability, &c.; and though these are usually believed to be generally absent, the patient's relatives often can presage an attack from the alteration in his manner and actions. Sometimes he is conscious himself of an approaching seizure.

Aura.—The above prodromata are not to be confused with the immediate warnings or auræ which usually come on a few seconds before the tonic spasm; in many cases the aura is entirely absent. The auræ are very varied, but a patient who has a well-marked warning generally experiences the same identical aura immediately preceding each attack. Its nature may be *sensory*, *motor*, *visceral*, or *psychical*. The aura is often best marked in mild seizures, and in rare instances it may be the only phenomenon, the impairment of consciousness being so slight as to be imperceptible in some attacks.

Sensory auræ are most common. Numbness, pain, sensation of spasm, tingling, &c., may be felt in some part of the limbs; or the special senses—sight, hearing, taste, and smelling, may be involved in the warning, causing sensations of colours or flashes of light or blindness, of sounds as whistles, bells, &c., or of unpleasant smells and tastes. The sensation of spasm beginning in a limb and travelling upwards, and the special sense-auræ, often afford valuable evidence of the site in the cortex where the discharge occurs.

Motor auræ may demonstrate themselves in complicated movements like running, turning round, vertigo-like motions, &c.; rarely do clonic spasms or twitchings of a particular part precede the general convulsions in idiopathic epilepsy, though these are the rule in the Jacksonian variety.

Visceral auræ are felt in the stomach and cardiac regions, though the pneumogastric and cephalic sensations, described as of a bursting feeling, are not rare.

Psychical auræ, as feelings of terror, hallucinations, joy, or anger, or of a vague dreamy stage, or the sudden recollection of some past unfamiliar event or place, may be experienced.

The seizure is often preceded by a loud cry or startling yell; the unconscious patient falls forcibly as if knocked down, and is often injured in the fall.

Three stages of the fit are usually described—viz. that of tonic spasm, clonic convulsions, and coma.

The *tonic* contraction affects all the muscles of the body; its rapid onset is what throws the patient off his feet and causes the laryngeal sound or cry. Though general, it usually involves one side more than the other, so that the head or even the entire body may be rotated; the arms are unequally affected, they are flexed at the elbow or extended, the wrist is flexed, the thumbs adducted, and the fists tightly clenched. Usually the lower extremities are rigidly extended. The mouth and eyes are turned generally to the side most strongly tetanised; the eyelids may be widely open or shut, and the pupils are dilated. The radial pulse can be felt with difficulty owing to the spasm of the muscles, though the carotids may be throbbing obviously.

The face, at first pallid, soon, owing to the fixation of the breathing muscles, becomes livid. Sometimes the tongue is caught between the rigid jaws and severely bitten; but this is more likely to occur in the following stage.

The duration of the tonic spasm varies from five or ten seconds, or less, to half a minute, during which time the breathing is entirely suspended.

The *clonic* stage begins in slight twitchings or tremulous movements, which rapidly develop into violent convulsions. The tonic spasm, as its name implies, consists in a *continuous* contraction, resembling that seen in tetanus, The essential feature in the clonic convulsion is the presence of a series of rapidly alternating contractions and relaxations or remissions. All the muscles may be involved, the movements of the facial muscles producing ghastly distortions. Lividity at first increases, but gradually lessens, owing to the admission of air to the chest. The tongue is often bitten at the side by coming between the snapping jaws; blood, saliva, or frothy mucus oozes from the lips. The conjunctiva does not react; the pupil is frequently dilated, but it usually oscillates between dilatation and contraction, and profound unconsciousness remains. The bladder and bowels may discharge their contents, sometimes semen is ejected, and during the violent contractions the shoulder-joint may be dislocated. Usually the convulsions cease as suddenly as they began; but in rare cases there is a gradual diminution in their severity before final cessation of movement.

The duration of this stage may last from a half to five minutes, after which there is a partial return to consciousness for a brief period, during which the patient may be roused; but he may without such interval pass directly into the final stage.

Usually there is profuse sweating present towards the end of the convulsion.

The stage of *coma* follows almost immediately upon the cessation of the movements; in it the consciousness is usually obliterated, though, as just stated, the patient may be roused for a moment. Its duration is most variable and difficult to determine, as it passes into deep and apparently natural sleep, which may last for one or two hours. Occasionally the stage of coma is absent, in which case headache may be severe; this may continue after sleep, and is sure to follow if the patient be roused up. The breathing may remain stertorous and the face somewhat livid; during the comatose period the pulse speedily falls to normal, after showing increased tension and frequency during the clonic period.

The reflexes are all absent during and for some time after the attack, but on their return the knee-jerks may be increased, and an ankle-clonus obtained. Vomiting is common after the coma; when it occurs earlier it may produce asphyxia. There is usually a copious secretion of urine, unless profuse perspiration has followed the clonic stage, and the first urine passed may contain albumin.

Transient paralysis from exhaustion of the centres, slight aphasia, and contraction of the visual field are often noticed. Ecchymoses or petechiæ into the skin or under the conjunctiva are as important as tongue-biting and expulsion of urine, since they assist in establishing the diagnosis in night attacks.

Of great importance is the mental condition after the attack. This will be referred to later on in connection with the minor attacks.

PETIT MAL

In this form or type of epilepsy there is a transient loss or impairment of consciousness without muscular spasm or clonic convulsion. The patient may feel any of the auræ already mentioned; but as a rule the attack begins suddenly without warning, and is over in a few seconds—often inside five seconds. There is rarely falling or cry, and should the patient be engaged in conversation all that is observable is a slight pallor, a fixed vacant stare, and a pause in speech, which is resumed in a few seconds as if nothing had happened. In taking his meals there may be a little awkwardness in the use of his knife, fork, or spoon, which is rarely dropped; but motion is suspended for a number of seconds, and with a dazed look he continues apparently unconscious that anything has occurred. The pupils are dilated, and the face usually flushes slightly at the end of the attack.

These attacks may constitute the only feature in the disease, and the patient may be unaware of his ailment. Sometimes

they alternate with severe convulsive attacks of grand mal, and it is remarkable that the aura may be better marked, though of the same kind as that which precedes the major attack, or it may be absent in the major and present in the minor attack.

The sensation experienced by the patient is one of fainting or of vertigo, or a colour-sensation may be felt; but all sorts of feelings are experienced, and in a small number of cases there is only some slight impairment not amounting to actual loss of consciousness. In women especially the urine may be expelled during the attack, but the writer thinks that the bladder is never emptied. A patient with repeated and constantly occurring spasmless seizures may occasionally exhibit tonic followed by clonic twitchings, especially about the mouth; such attacks were placed in a separate group by Reynolds as *epilepsia mitior* with evident spasms, when these latter were usually present.

The *post-epileptic state* is of great importance, as automatic movements of a complex nature may follow; these are much more common after minor attacks than after the major seizures. Thus, after the transient unconsciousness the patient in a dazed condition may start to run about, to undress himself, or to expose his genitals, or to steal any articles within his reach. Of these acts, which may occupy several minutes, he has no knowledge after coming to his senses. Some authorities, as Oppenheim, regard these acts when occurring independently of all seizures as *psychical equivalents* of the epileptic attacks; others describe them as instances of *masked* or *psychical epilepsy* or *epilepsia larvata*. Delirium associated with maniacal excitement may follow mild attacks, and it may last for many minutes, during which homicidal acts of a serious nature, which have all the appearance of purpose, may be perpetrated. The condition is different from the insanity which often is associated with epilepsy, and which shows itself in the intervals between the attacks. Hysterical manifestations may show themselves immediately after the seizure is over, and at no other time, in both men and women; these constitute the post-epileptic hysterical convulsion of Gowers, so liable to be mistaken for simple hysteria or *hystero-epilepsy*.

Reflex epilepsy is sometimes described as a special variety; it is seen in those cases of peripheral irritation following old injuries which leave a sensitive scar behind them. For a long time the convulsion may be confined to the injured limb, but ultimately it eventuates in the idiopathic form, becomes general, and persists after the removal of the cause; the same may be said of the convulsions sometimes caused by tape-worm.

Organic brain-lesions as hemiplegia, tumour, &c., and injuries to the cortex producing Jacksonian epilepsy, referred to

upon page 195, may long be confined in their manifestations to the regions associated with the particular part of the cortex involved, and should not be included in this section. But in all these conditions the area of instability is liable to extend, and ultimately the cases may pass into idiopathic epilepsy; though the convulsions may begin in the old site, they become bilateral and general. The truth of this is obvious in the usual result—viz. the permanence of the general convulsive attacks after the removal of the original local cause when this is of long-standing.

Course and Duration.—The fits obey no known law as regards their frequency or severity: they may occur two or three times during a year, or as many times every day, for long periods. The most common form of beginning is in the severe or grand-mal attack, which is not repeated for some months afterwards; but the general tendency is for the attacks to occur gradually or irregularly at shorter intervals. Possibly the first severe attack may have been preceded by minor attacks which had not been recognised for years. Sometimes they only occur at night, whilst in some patients they always appear during the waking hours. Minor and major attacks may alternate, or the type may remain true; frequently the minor passes into the major variety. Often there may be a long interval, and then a series of seizures may rapidly follow each other, with a long interval between the next series.

The *status epilepticus* is said to occur when a series of fits follow in such rapid succession that a new one comes on before the old one passes away; and this may continue for days, when a very serious condition of profound coma, with exhaustion and high temperature, or hyperpyrexia, is developed, which often ends fatally. (The temperature in the ordinary attacks is often normal, seldom raised by 1° F.)

Usually at menstruation the number of attacks are increased, or they may only appear then; but sometimes they disappear during pregnancy.

In some cases a long absence of attacks has been noticed to produce a change in the patient's manner and spirits, which are much depressed; this usually passes off after an attack.

The *mental condition* in epilepsy is a point of vital importance. Already the transient mental condition immediately succeeding the attack has been referred to, but the condition of the epileptic's mind between the attacks is another matter. In a small percentage of cases an acute attack of insanity may supervene after a rapid series of epileptic seizures, and it may become permanent; but the rule is that gradual mental deterioration sets in.

According to the age of the patient when the disease sets in there will be idiocy or imbecility, and it is hard to determine

how much of the feebleness of mind in these cases is caused by the epilepsy or is to be attributed to the congenital or hereditary general neurotic condition. Many, perhaps the majority, of epileptics never show any marked mental deterioration which would justify their being classed as insane, but the great majority of all epileptics exhibit some mental weakness, as loss of memory, irritability, or impulsiveness. Between this and dementia, all forms and grades of mental failure are noticeable. The often-quoted examples of Napoleon and Julius Cæsar should be regarded as the brilliant exceptions which prove the general rule that epilepsy is but rarely associated with the highest type of cerebral development.

The recent figures of Turner, based on the Chalfont St. Peter colony, show that 13·6 per cent. of the cases could be regarded as intellectually normal; and this proportion practically corresponds with the figures from the returns of the Stettin Institute. Mental deterioration is likely to occur in proportion to the earliness of age at the onset, and to the length of the duration and severity of the fits. In major epilepsy the danger of mental weakness *is less than in petit mal*; but the largest number of cases of severe mental failure is to be met with where both types of attack co-exist. Epileptics are found to develop a characteristic physiognomy to which the name *facies epileptica* has been applied; it is not confined to those with mental symptoms, though it is best marked in them. Turner found it present in 45 per cent. of all cases. It has been described by him as a peculiar expression of heaviness and dulness with an absence of emotional mobility of the features. The majority of epileptics are religious enthusiasts, and in those showing mental symptoms there is often most marked perversion in this respect.

Diagnosis.—With day attacks of the major form there can be little difficulty, when *uræmia* is excluded by the condition of the urine and *lead-poisoning* by its history and paralytic signs.

Hysteria is more difficult of exclusion, but the absence of true epileptic aura and of a sudden loss of consciousness rapidly followed by a *tonic*, quickly followed by a series of clonic spasms, the absence of tongue-biting, of dilatation of the pupil, and of involuntary micturition, stamp the hysterical affection. This, moreover, has a very much longer duration of fit, with irregular movements and struggling, and often shouting or talking, which seldom ends in the coma or deep sleep which usually follows epilepsy. A recently recognised differential point is of value: Babinski's toe-phenomenon is present before and after the seizure of epilepsy, and never in hysteria; the absence of the pupillary reflex is strongly in favour of epilepsy.

From *organic brain-disease* epilepsy is generally differentiated by the local nature of the spasm at the beginning of the seizure in the organic condition, and by such signs of cerebral disease, as eye or other local paralyses, severe headache, vomiting, and optic neuritis.

General paralysis of the insane may be complicated by convulsions, but the tremors about the lips, inequality of the pupils, and grandiose symptoms are distinctive.

Malingering is common and is easily recognised by being generally over-done, and the presence of the pupillary and conjunctival reflexes is conclusive.

The *convulsions of childhood* which usher in acute fevers, and those which are produced by intestinal parasites, rickets, dentition, and other reflex causes, are not to be distinguished from idiopathic epilepsy except by their disappearance on the removal of the exciting cause ; when they remain they must be regarded as idiopathic epilepsy.

Petit-mal attacks are often mistaken for cardiac *syncope*. The chief points in the diagnosis are the suddenness of the loss of consciousness and the rapidity with which the patient recovers his senses in minor epilepsy ; the pulse may disappear in syncope, and there is little mental confusion afterwards and never any trace of local spasms about the mouth, never involuntary micturition or any automatic movements or psychical disturbances, and deep sleep rarely follows.

Menière's vertigo may simulate petit mal, but total unconsciousness is not common ; the vertigo remains severe after the other features of the attack pass off, and there is tinnitus and some deafness and vertigo in the intervals. These symptoms are absent in the group described as *epileptic vertigo*, in which a sensation of vertigo is the usual warning or aura of a true epileptic seizure. Gowers has reported several cases where both affections co-existed.

Nocturnal attacks may be unrecognised for a long period, but the tongue-biting and bed-wetting are almost conclusive evidence, and search should be made for hæmorrhages under the skin or conjunctiva.

Prognosis.—A fatal termination of a fit is very rare, unless resulting from some injury caused by the fall or by suffocation should the patient turn over on his face in bed or should vomited matter block his windpipe. The status epilepticus is very rare and not necessarily fatal, about half the cases recovering from this formidable complication. As regards permanent cure, the prospects are not bright; taking all cases of confirmed idiopathic epilepsy together, it may be safely predicted that not more than one case in ten will permanently recover from the disease. Heredity, strange as it may seem, does not appear to lessen the chance of recovery. Recovery is very rare in cases where the disease began in the

first decade, and amongst those starting between the ages of fifteen and twenty one-third may be expected to become arrested. In subjects between the ages of twenty-five and thirty years at the commencement the prospects of cure, according to Turner, are at their lowest, but they gradually continue to brighten as the onset of the disease has become later. The duration of the malady, when the prognosis has to be given, is important; it becomes less hopeful in proportion to the length of the duration, but recovery has been known to follow in cases where the disease had existed for a quarter of a century. The frequency of the attacks diminishes the hope; the prospects of recovery are always worse in the minor form than in the major.

Those cases which rapidly respond to bromides are hopeful, and night cases are more promising than day cases.

The prognosis as regards mental deterioration is referred to upon page 392.

Though marriage does not usually influence the prognosis, epileptics should be forbidden to marry.

Treatment.—In the author's 'Dictionary of Treatment' (4th ed.), pp. 278–286, almost every drug used for epilepsy is discussed, with all other details of treatment; only a few general principles may be here referred to.

In cases seen early, it is of vital importance to search out for any possible reflex cause, as dental, ocular, nasal, aural, intestinal, or genital irritation, or the presence of scar-tissue, especially if adherent to bone. These should be at once removed. Every health law should be most implicitly obeyed, and all mental worry avoided; a vegetarian or purin-free diet is most desirable, especially in heavy eaters, and Purves Stewart has drawn attention to the great value of a diet free from sodium chloride. The patient must be warned against the dangers of placing himself in any position where a fall might lead to serious consequences.

Where there is a well-marked local aura, sometimes the fit may be prevented by tying a temporary ligature above the site of sensation; by strongly pinching the region; or by forcible flexion or extension of the limb. Nitrite of amyl inhalation, in a small percentage of cases, is sufficient to ward off a fit, but it must be remembered that often the patient feels ill for days after the prevention or abortion of an attack.

During the convulsion it is useless to attempt any restriction of the movements; the physician or attendant must be content with taking such measures as will prevent the patient causing injury to himself by tongue-biting or striking his head against hard objects or drawing vomited matters into his larynx, and all restrictions about the neck should be promptly removed. He should be permitted to sleep after the cessation of the convulsions. In the status epilepticus, cold

sponging and ice may be freely used, and large doses of bromides with chloral should be administered per rectum.

In the intervals between the attacks the bromides afford almost the only hope of cure or amelioration. Experience has proved that one salt of bromine is practically as good as another; but when large doses are necessary for long periods the sodium bromide is the least depressing. A good rule is to begin with three daily doses of 20 grs. each, which may be increased after a few months to 30 grs. if marked diminution in the frequency of the attacks be not produced. Some authorities recommend much larger quantities, and advise the drug to be pushed till the full physiological action is observed —viz. drowsiness and loss of the palate-reflex.

In all cases the remedy should be given in a state of free dilution, and its administration must be continued for a period of years; even when the fits have entirely ceased, it should be rigidly enforced for a period of eighteen months or two years before marked diminution of the dose be attempted. Bromism may call for a lessening of the dose, or for its temporary suspension, but such an event should always be anticipated.

Acne is lessened or prevented by the addition of arsenic, but this drug should be stopped for considerable periods during the treatment. When the attacks only occur during the night, one large dose (1 dr.) may be given an hour before bedtime, and half this amount about noon.

The aim of the physician should be to gradually push the drug till he becomes satisfied that an impression has been made upon the disease, after which the dose can be permanently fixed; this can only be done accurately by the patient's friends keeping a correct chart with the number and dates of all the seizures carefully marked down from time to time for long periods.

One great difficulty in carrying out the bromide treatment in private practice is the idea which seizes upon the minds of the patient's friends in those cases where mental deterioration becomes marked: they often attribute this to the drug, and refuse to permit its continuance.

Innumerable drugs have been tried where the bromides fail, as they often do, and belladonna or atropine sometimes succeeds in these cases. Some authorities always combine atropine with the bromides; the drug should be given in all cases during the temporary suspension of the bromine-treatment. Flechsig's method of combining large doses of opium with the bromides is not to be recommended. Bromipin or brominol, and ethylene bromide have proved useful substitutes in some cases for the ordinary salts.

Nitroglycerin often relieves petit mal; eight to ten tabloids may be taken in the day, or the nitrite of soda may be substituted.

Borax is placed second to the bromides by Gowers, but it often upsets digestion, and may produce dermatitis.

Salts of lead, gold, silver, copper, zinc, and nickel have been vaunted, but they are gradually becoming only of historic interest in the literature of the therapeutics of epilepsy.

The surgical treatments of idiopathic epilepsy by ligature of the carotid or vertebral arteries and the extirpation of the chief ganglion of the cervical sympathetic, as carried out by Alexander, are measures of such gravity that they cannot be recommended. The same must be said of the procedure of excising that portion of the motor cortex which controls a region in which a well-marked local aura has been constantly felt in idiopathic epilepsy.

Operative treatment for traumatic epilepsy produced by the irritation of depressed bone is justifiable ; if pursued at an early stage before the Jacksonian convulsion or partial epilepsy has become general, there is a fair prospect of cure by trephining and removal of scars, splinters, and depressed bone.

Operative measures are usually worthless where the trauma is of old standing, as then consciousness is lost early in the attack, and the epilepsy has probably become general or identical clinically with the true or idiopathic form, and will remain after the removal of the original exciting cause.

Occasionally the scar caused by an old cranial injury becomes an epileptogenous zone—*i.e.* it is painful, and pressure upon it will bring on a convulsion ; under these circumstances trephining becomes justifiable, even though the convulsions are general, as the case may prove to be one of reflex epilepsy. Abscesses, tumours, or cysts producing Jacksonian convulsions should be dealt with by a free trephine-opening.

EPISTAXIS

This is the name given to hæmorrhage coming from the interior of the nose.

Etiology.—Bleeding from the nostril is often a local surgical symptom caused by rhinitis, ulceration, syphilis, cancer, trauma, caries, polypi, &c., in which cases the site of the bleeding vessel will be determined by the local lesion. Constitutional causes may produce it, and these are numerous and sometimes obscure. It may usher in or arise during the progress of the exanthemata, and it is a diagnostic sign of value in the early stage of typhoid. Malaria, relapsing fever, erysipelas, plethora, grippe, diphtheria, and jaundice sometimes have this symptom a prominent one in their clinical picture. Chronic anæmia caused by leukæmia, purpura, scurvy, chlorosis, &c., frequently produces severe epistaxis. Bright's disease and atheroma, cirrhosis of the liver, and

pulmonary diseases, as acute bronchitis, emphysema, and pertussis, often produce it. It may be an early symptom in chronic valvular disease, when compensation begins to fail, especially in mitral stenosis.

It is common in young subjects before or about the age of puberty, and it is constantly witnessed in mountaineering and balloon ascents; and many cases are reported where its appearance indicated that it was a form of vicarious menstruation. It may result from the action of drugs, as seen in phosphorus-poisoning, and after long courses of the salicylates, or from breathing very hot or very cold air.

Diagnosis.—When the cause of the epistaxis is the impoverished state of the blood, the fluid may ooze from both nostrils; though this is rare. Both in the constitutional as well as in the local cases resulting from an abnormal state of the mucous membrane, the bleeding-point is usually situated upon the anterior and lower part of the septum, where the mucous surface may be often seen to be spongy, varicose, or ulcerated, and this spot should always be sought for. In either case, as the patient lies upon his back, the blood may trickle down the pharynx, and after reaching the stomach may be vomited, simulating hæmatemesis, or it may be coughed up from the pharynx and be mistaken for hæmoptysis.

The amount varies from a quantity so small as to cause no feeling of apprehension, to several pounds of blood, inducing a rapidly serious anæmia, which may cause fatal syncope; though death is upon the whole a rare event.

Prognosis.—This is always serious when the attack is the result of an anæmia of long-standing, and when occurring in the late stage of an affection like leukæmia a grave prognosis is obvious. The recurring attacks in subjects of cirrhotic kidney, with atheromatous arteries, are apt to be followed by an attack of cerebral hæmorrhage at a later stage, some time after all epistaxis has ceased.

In young subjects the prognosis is, as a rule, always favourable, though the amount of hæmorrhage may be large.

Treatment.—In many instances met with amongst plethoric subjects the hæmorrhage should not be interfered with unless it be profuse, and this is especially true in those cases preceded by severe congestive headaches. A smart saline purge may relieve both symptoms should there be any apprehension on the part of the patient. In subjects labouring under renal cirrhosis and atheroma, with a high-tension pulse, it is just possible that the epistaxis may have averted a cerebral hæmorrhage, and active interference here is also undesirable.

When the amount of hæmorrhage justifies interference, the old-fashioned simple plan of placing the patient upon his back, with his head well raised and his arms elevated above his head, with cold compresses on the temples and occiput,

may be tried. If this fail, he may be placed in a high arm-chair, in the sitting posture, with his legs immersed in a deep pail of very hot water, to which a little mustard has been added.

Where the bleeding continues profusely the nostril must be plugged anteriorly by means of a speculum and cotton-wool or lint smeared over with iodoform or a tannin preparation, after which gentle pressure by the fingers from without will control the hæmorrhage. The writer's plan of inserting a large plug of the lycoperdon giganteum meets every indication: it stops the hæmorrhage instantly in every case, unless it be coming from far back in the posterior nares, and the plug may be permitted to come away spontaneously, which it will do in a few days; it is, however, not aseptic. Hot or very cold water alone, or bearing astringents in solution, is recommended; as a rule, they are unsatisfactory, and strong iron solution should only be employed to touch a visible bleeding-point.

Adrenalin solution is the most cleanly and at the same time the most reliable of all local astringents; it will probably replace the formidable operation of plugging both nostrils from the posterior nares, and it renders unnecessary the various forms of rubber bags which require inflation after being inserted. Chloride of calcium internally is of the greatest value in all serious cases, but ergot, tannin, and other so-called internal astringents are worthless.

In the recurring nose-bleeding, the ulcerated spot on the anterior portion of the septum should be treated by the galvano-cautery, or cauterised with chromic acid.

It is unnecessary to say that the primary condition causing the epistaxis should be sought for and treated, but in a considerable percentage of cases no cause can be found.

ERGOTISM

Ergot contains three active substances—viz. (*a*) *ergotinic acid*, a powerful local irritant which induces suppuration when a fluid preparation of the drug is injected hypodermically: it is a depressant to the central nervous system; (*b*) *sphacelinic acid* (which contains sphacelotoxin), a resinous substance devoid of nitrogen, which has the power of producing dry gangrene by constricting the arteries, as seen in the comb of the cock after injection of the drug; it increases blood-pressure by the vascular constriction, and it augments the reflex activity of the cord; (*c*) *cornutine*, an alkaloid which has a powerful selective action upon the involuntary muscular tissue of the uterus and intestines, as well as a powerful stimulant action upon the medulla and vaso-motor centres.

When grain affected with the ergot-fungus has been used for any length of time as a food, certain symptoms are observable in those consuming it. These are caused by a combination of the actions of the active principles of the drug. In past times epidemics of chronic ergot-poisoning were very common in Russia and some other European regions. The symptoms may be grouped into two definite types, both of which may be seen in the same epidemic; but as a rule the *gangrenous* type predominates in one epidemic, whilst the *convulsive* form is the most obvious in another.

The gangrene is caused by the sphacelotoxin of the sphacelinic acid; it usually affects the fingers and toes, but occasionally an entire limb may become cold, black, and anæsthetic, and shrivel up and drop off without hæmorrhage, owing to the occlusion of its arteries by muscular constriction, followed by hyaline thrombosis. Internal organs may suffer in a similar way. The symptoms caused by nervous disturbance first show themselves in weakness, depression, headache, vertigo, and itching of the skin, followed in a few weeks by severe convulsive seizures, often of a typical epileptiform type. In milder cases the seizure, consists of a severe prolonged cramp or tonic spasm, or this may follow the clonic convulsion, and end in contractures flexing the upper and extending the lower extremities.

Mental symptoms are commonly present, and in very chronic cases may end in dementia. The posterior columns of the cord become affected, and as a spastic paraplegia with ataxia is characteristic of *pellagra* (a disease caused by eating decomposing maize), so in chronic ergotism the typical spinal lesion produces symptoms closely resembling those seen in locomotor ataxia.

The condition was often fatal before its causation was understood. The prognosis is good if the symptoms are recognised early and the food-supply changed immediately; the obvious treatment being to stop the use of the fungus-poisoned flour.

ERYSIPELAS

This is an acute contagious spreading inflammation of the skin or of a mucous membrane, caused by the presence in the lymph-spaces of the streptococcus pyogenes.

Etiology and Pathology.—As seen in the definition just given, the disease is the result of infection with the specific micro-organism, but the causes which lead to the increased virulence of the coccus necessary for the production of erysipelas are unknown. The parasite does not enter the blood-vessels, according to Miles, but rapidly develops in the lymph-spaces in the spreading margin of the inflamed patches, and invades

the upper layers of the skin ; the toxin which it manufactures is absorbed, and produces the severe fever and constitutional symptoms characteristic of the disease.

Though a so-called *idiopathic* form of the disease has long been recognised, it is probable that a wound or abrasion always is present, affording a channel for inoculation.

The disease is very liable to attack open wounds, and formerly was very common in the surgical wards before the introduction of aseptic and antiseptic methods. The coccus flourishes in the presence of overcrowding and bad ventilation, and clings for a time to bedclothes and furniture, and to the hands of nurses and attendants. Puerperal patients, alcoholics, infants, and enfeebled aged folk are readily affected by it, and in some apparently healthy individuals there is a marked susceptibility, as shown by frequent recurrences, which also prove that immunity is very short-lived ; it is most prevalent in the early spring.

The streptococcus does not usually produce suppuration. When this occurs, probably other pus-forming organisms are acting in conjunction with it, and these are the cause of the dreaded deep suppurative cellulitis which occasionally complicates the disease, and which may end in gangrene or cause pyæmia, pulmonary or other infarcts, ulcerative endocarditis, metastatic abscesses, pneumonia, suppurative meningitis, pericarditis, empyema, &c.

The changes found after death correspond with the symptoms observed during life ; subcutaneous abscesses are frequently observed, and the lymphatic glands are invariably affected in the vicinity of the diseased skin. The blood is found to be almost black in colour and to have lost its coagulability, and the internal organs are deeply congested and purulent foci are commonly found in them, as in pyæmia and other septic conditions.

Symptoms.—The incubation of erysipelas is short—it is usually stated as varying from three to seven days ; but in the case of open wounds the writer has witnessed in the surgical wards, in the pre-Listerian days, an incubation of only twenty-four hours in several cases. Fehleisen found by inoculating the parasite for the cure of tumours, &c., that the incubation-period varied from fifteen to sixty-one hours.

The head or face is the usual site in cases seen occurring independently of surgical wounds, the virus effecting its entrance through some abrasion about the margins of the nostrils.

The first symptom is a feeling of malaise associated with chilliness and not seldom ushered in with a smart rigor, in which the pyrexia may rapidly rise to 102°–105°, with severe headache, backache, and vomiting.

At the period of invasion, as marked by the constitutional symptoms, or a few hours after, some redness appears about

the site of the abrasion, or upon the alæ or bridge of the nose, or on the cheeks. The blush of colour rapidly and irregularly spreads over the face and usually invades the scalp; it may be of a bright scarlatiniform hue or of a dull livid colour, or inflamed patches may appear, which rapidly coalesce. The inflamed area is tender to the touch, and the patient complains of a severe burning or smarting sensation associated with a feeling of tension; it is raised above the level of the healthy surrounding skin, and œdema soon becomes obvious on gentle pressure applied to the smooth, glossy, tense surface. The œdema is greatest about the eyelids, which soon completely close, and the lips swell greatly. When the ears are involved, the swelling causes intense pain and discomfort over the auricle, and the same result may be severely felt over the nasal cartilages. Blebs or bullæ form over the cheeks or forehead, from which an amber-coloured serum is obtainable, in which the cocci may be demonstrated.

The margins of advancing redness are raised and well defined, but a little beyond them there is marked tenderness, and the deep lymphatic vessels may be felt to be indurated under the normal skin. Though the inflammation may involve the entire face and scalp, causing great swelling and distortion of the features, it seldom extends beyond the chin to the anterior surface of the neck, and it usually stops at the margin of the hair below the occiput, though the cervical glands always show some enlargement and tenderness.

The inflamed area first affected may be showing signs of fading or resolution, whilst the margins of the inflammation are steadily and actively pushing out into the healthy skin of the forehead or scalp. A form of the disease is recognised in which the eruption disappears from the face and wanders from one region to another of the body, in its migrations involving a considerable portion of the skin; it is known as *erysipelas migrans*.

Whilst the inflamed area on the face and scalp is active the temperature usually keeps high, with slight remissions for several days—four to eight, or longer, in severe cases—and it generally falls more or less abruptly by crisis. The local temperature in the inflamed area is always markedly increased. The pulse is full and strong for the first couple of days, but in alcoholic subjects and in old people it soon exhibits marked signs of feebleness. The tongue is moist and the breath foul in most cases, but in the alcoholic and debilitated it soon becomes dry and brown; formidable delirium, and sometimes hiccough, and a typical typhoid state accompanies the change. There is always marked leucocytosis present, and the spleen is sometimes found to be enlarged.

A marked increase in the fever and pulse-rate, with delirium, restlessness, and vomiting, may indicate the supervention of

meningitis or sinus-thrombosis, and a succession of rigors may be the first warning of abscesses having formed beneath the scalp or in some distant part. It is necessary to raise the œdematous eyelids and watch the condition of the eyeball, which may sometimes be destroyed by the tension of an orbital abscess, if not promptly incised. The inflammation may extend to the middle ear, or it may involve the pharynx ; faucial erysipelas is a grave complication owing to the danger of œdema of the larynx.

The severity and duration of the disease vary greatly ; in many cases a superficial erythematous redness only marks its presence, and this may remain strictly localised, the fever and constitutional symptoms passing away in two or three days. In other cases, especially in the debilitated, the symptoms may be severe and the fever may continue for as many weeks, causing death from exhaustion or some complication.

The inflammation gradually, in moderately severe cases, resolves without leaving any evidence of its previous existence, save desquamation, though sometimes thickening and œdema may persist for a long time.

Complications. — Most of these have been incidentally mentioned, as meningitis, ear, eye, and throat affections. Albuminuria is often witnessed ; pneumonia and ulcerative endocarditis are formidable, and usually terminate fatally. Deep suppurative cellulitis, caused by the introduction of pus-forming organisms, may end in the destruction of large tracts of tissue, and true pyæmia may result. Puerperal septicæmia may have its origin in a mild superficial erysipelas ; hence the accoucheur should never attend lying-in cases after being in contact with erysipelas.

Of less consequence are the local multiple abscesses which often form in the affected area during the active stages of the disease or after all inflammatory signs have passed away. They should be promptly incised as soon as they appear.

Diagnosis.—This is as a rule easy, owing to the abruptness of the onset, with smart fever and the circumscribed infiltrated margin of the extending redness, with the circle of tenderness in the uncoloured skin outside all redness and œdema.

Acute eczema and *erythema* never present the well-marked constitutional symptoms of erysipelas. In the former the itching and local increase of temperature may simulate erysipelas, but the surface is always rough and desquamating, and the margins of the diseased patches are not so circumscribed ; and this is true of *acute dermatitis* also. *Eczema rubrum* of the legs is always chronic, and there is the presence of profuse general oozing of serous discharge.

Erythema nodosum simulates erysipelas, but the site of the raised swollen nodes on the shins and extensor aspect of the forearm, and its bilateral distribution, are distinctive.

Acute osteoperiostitis or *osteomyelitis* of a long bone may be suddenly ushered in with severe constitutional disturbance: deep-seated swelling and pain, followed *afterwards* by superficial redness, which, however, never presents the characteristic defined margin and superficial tenderness of erysipelas.

Herpes zoster affecting the Gasserian ganglion, has been mistaken for erysipelas, but the peculiar one-sided distribution of the eruption, following the course of the first, and more rarely of the second and third divisions of the trigeminal, with its grouped clusters of vesicles on an erythematous base, should be absolutely distinctive.

Lymphangitis is readily distinguished by the course of the redness, which follows the direction of the lymphatic vessels of the limb, whilst the spread of erysipelas does not follow in the direction of the lymphatic trunks.

Acute, diffuse, or *suppurative cellulitis*, though allied in its pathology to cutaneous erysipelas, is generally easily differentiated by its deep origin, which causes brawny induration and œdema *before* cutaneous redness occurs. When this appears, which is often only after the advent of deep sloughing, the redness is livid and not sharply circumscribed, and not easily made to disappear by pressure contrasting with the bright glossy hue of the tense skin in erysipelas.

Prognosis.—In simple uncomplicated cases this is generally good—in healthy subjects perhaps the all-round mortality does not exceed 5 per cent. In debilitated alcoholic or aged patients the forecast must be always serious to grave, and this is true also as regards patients suffering from Bright's disease and those in the puerperal state. The presence of complications deepens the gravity of the prognosis in proportion to the importance of the organs or parts involved, local abscesses being of little moment compared with the evidence of deep pyæmic suppuration in distant parts. The prognosis is usually very serious when the bullæ contain deeply blood-stained liquid instead of clear serum. Umbilical erysipelas in newly born infants is nearly always fatal. The gravity of the case bears but slight relation to the extent of surface involved in many instances. Profuse diarrhœa is generally a serious symptom. Death may occur from the exhaustion produced by the continuous high fever in protracted cases, from toxæmia, or from any of the more serious complications.

Treatment.—The details and lists of remedial agents are discussed at length in the author's 'Dictionary of Treatment,' pp. 288–294. Only the general principles need be here summarised.

Rigid isolation and thorough disinfection to prevent the spread of the disease are imperative.

Mild cases of erysipelas would probably get well without any treatment, but the old-fashioned method of pencilling the margins of the redness with strong caustic solution, or the more modern plan of Miles, of painting on several coats of strong iodine solution beyond the margin, in order to check the spread, should always be resorted to.

Of local applications there is no end. The best for routine use is ichthyol, made into an ointment with an equal amount of lanolin and smeared freely over the face, which should be covered by a lint mask. Mild cases get well rapidly under any bland dusting-powder, as starch or flour, with zinc oxide.

Internal remedies are indicated by the presence of complications and by the previous condition of the patient and his habits as regards alcohol. As a routine drug, iron has held its own against all competitors ; large doses of the tincture probably aid phagocytosis, which is nature's method of cutting short the disorder.

ERYTHEMA

This term is often loosely applied to all conditions of the skin associated with marked redness, and some authorities include even the rashes of scarlatina, &c., under erythema.

The name should be restricted (1) to those skin-affections whose chief clinical feature is redness caused by a simple hyperæmia or congestion ; and (2) to those where the hyperæmia passes into inflammation and exudation.

Amongst the first or congestive group is included the following *generalised* form of erythema:

E. roseola—a diffuse redness or rash consisting of large macules which is most frequently met with in young children, and which is ushered in sometimes with feverishness and malaise. Its chief interest lies in the difficulty of distinguishing it from measles, rötheln, and scarlatina. It is identical with *E. scarlatiniforme*, which is the name given to it when it is supposed to be not idiopathic, but a symptom of some apparent toxic condition, as, for example, when it follows the internal administration of drugs like quinine, boric acid, salicylates, belladonna, &c., or the sera used in diphtheria, tetanus, &c., and when it is found in association with rheumatism, typhoid fever, and various septic conditions ; its macules are usually punctiform. From *scarlatina* it may be diagnosed by the absence of the typical condition of the tongue and throat (though there may be simple redness of the fauces) and by the mild type of the fever ; desquamation may follow it, but this sets in earlier than in scarlatina. The chief diagnostic point lies in the fact that the eruption is

rarely if ever universal; the borders of the erythema are more or less circumscribed, and regions of healthy skin may be observed between the erythematous areæ.

Rötheln and *measles* are excluded, the first by the absence of the enlarged glands, and the second by the coryza.

Localised forms of erythema of the simple congestive type are numerous.

E. pernio, or chilblain—the congestion of the skin over the hands and feet which occurs in cold, damp wintry weather amongst young subjects with feeble circulation.

E. ab igne is the name given to the hieroglyphic markings which occur on the shins of patients who habitually toast their legs before an open fire. *E. caloricum* is the localised redness which results from exposure to heat-rays from the sun, from hot water, electricity, &c.; whilst some writers include under *E. solare* the congestion which is caused by light-rays of various kinds.

E. intertrigo, already referred to under 'Eczema,' is the erythematous redness which appears when two folds of skin lie in contact, as under the breasts of fat women, between the thighs and about the buttocks of children; the retained secretion of the skin is apt to soon induce a true eczema. It is identical with the rash which is caused by the urine-soaked napkins of infants, and which is styled *E. gluteale*.

E. venenatum is the name usually given to the congestion caused by irritant drugs coming into direct contact with the skin, as mustard, arnica, rhus, primula obconica, chrysarobin, blue ointment, sulphur, arsenic, and a host of other drugs; but as the irritation is prone to pass beyond the congestive stage, Crocker and others class this form as a true dermatitis or inflammation, and the same remark applies to *X-ray erythema*, which, when severe, passes into *X-ray dermatitis*, and the *E. caloricum*, which may eventuate in severe inflammation.

E. læve is an important variety represented by the congestive redness which appears often in dropsical limbs, and which if neglected often leads to sloughing or ulceration.

E. paratrimma represents the early stage of bed-sores, being the local congestion which follows pressure or friction over bony prominences.

E. fugax is the name given to the fleeting erythematous redness which suddenly appears in patches over the nose and cheeks in transient dyspeptic conditions, and in certain fevers, and in some persons upon taking wine even in small quantities.

The second group of erythemas, where exudation is the chief feature, includes the well-recognised form of affection known as

ERYTHEMA MULTIFORME

or E. exudativum multiforme, which is closely allied with E. nodosum and E. iris, and Peliosis rheumatica.

The term *E. multiforme* is often applied to the entire group of exudative erythemas, and the following are regarded as simple varieties or different types of the eruption—*i.e. E. papulatum, E. annulatum, E.* or *Herpes iris, E. nodosum.* Hebra was the first to show that all these differently designated skin-affections were but different manifestations of one morbid blood-condition which might be brought about by different causes.

Symptoms.—The clinical picture varies greatly in different cases, but in these variations lies the essential feature of the affection as expressed in the term 'multiform.'

The lesions which constitute the eruption appear usually after a smart febrile attack, often with joint-pains, sore-throat, and chills, or these may not appear till the eruption comes out; but sometimes a typical case may be unassociated with any fever or malaise, and occasionally acute rheumatism and cardiac complications may appear at a later stage.

The site of the eruption is usually the dorsum of the hands and feet, the face and limbs, but often the trunk is involved. There is redness from bright scarlet to lividity, and the lesions are fairly symmetrical. They consist most frequently of papules of a deep red colour, which almost disappears on pressure at first; these come out in groups or clusters and arrange themselves in variously shaped and sized patches or plaques. The individual papules on their first appearance are usually about the size of a split pea, but they increase in size after some hours. If they remain discrete the name usually given to the lesion at this stage is *E. papulatum*; when the individual discrete papules enlarge, *E. tuberculatum* will signify the appearance of the lesion, each elevation of which may become as large as a split cherry, or the papule may develop into a node of the dimensions of a small poached egg—*E. nodosum.*

After a time, as the papule or tubercle enlarges at its periphery it wastes in the centre, which becomes depressed, producing an umbilicated form, and the raised circumferential part remaining a deep red gives a ringed appearance to the lesion—*E. circinatum* or *annulatum.* Sometimes several concentric rings of varied tints of redness surround the elevation, producing the condition known as *E. iris.* In rare cases the papule becomes a vesicle, surrounded by a depressed violet zone enclosed by a white ring with a pink margin—*Herpes iris.*

When the primary papules, tubercles, vesicles, or rings coalesce with neighbouring ones, still more varied lesions result from their expansion, forming larger rings—*E. marginatum*; or producing irregularly broken circles—*E. figuratum* or *gyratum*, in which the eruption is fading in the central parts.

Many of these forms may be seen in the same case at a given moment, as the eruption comes out in successive crops; but in

any particular case the type of the eruption may remain papular, herpetiform, ringed, or marginate, but seldom will two cases be seen alike. In recurrent attacks the same type is usually preserved which predominated in the first seizure.

Grafted upon any of these forms, *bullæ* may appear, and even suppuration or gangrene may occur, simulating pemphigus, when the inflammatory action is intense.

Though in all severe cases there is more or less effusion of blood-elements, as is seen by the staining or pigmentation left behind, in one type of the affection true hæmorrhages are the constant rule. This form is known as *Peliosis rheumatica* or *Purpura rheumatica*, or Schönlein's disease. As implied by its double names, this variety is closely associated with acute rheumatism on the one hand, and with purpura on the other. In addition to the erythematous papules, there are true extravasations of blood producing petechiæ which do not fade on pressure. These come out on the legs or in the neighbourhood of the joints which are the seat of the acute articular inflammation. In some cases the articular manifestations may be slight, or even unnoticed, but endocarditis may show itself. The writer has seen this affection develop between the attacks of acute rheumatism, obviously taking the place of an attack, though the joint-pains were absent. When wheals appear with the purpuric spots, the condition is described as *Purpura urticans*.

The duration of the eruption in multiforme is not easily defined, as it fades in one place whilst young and active in other regions, The entire attack, however, may be said to usually last from fifteen to thirty days, though in rare cases successive crops of the eruption may continue for months. A considerable amount of staining is left behind which may last for a long period. Recurrences are common ; they are especially liable to happen in the spring months ; no age is exempt. Complications are met with in a considerable percentage of cases. Joint-troubles have already been referred to. Diarrhœa, vomiting, endocarditis, pleuritis, and pneumonia have been observed. In one of the writer's cases the eruption occurred first on the conjunctiva, producing severe irritation. As a rule, there is little itching, except in *E. iris*. Sore-throat often accompanies or precedes the eruption, and this is commonly the case in peliosis rheumatica.

Pathology and Etiology. — The morbid anatomy of erythema multiforme consists of the usual changes characteristic of inflammation. There is exudation and cell-proliferation round the dilated vascular walls in the corium, with some hæmorrhage. Unna and others maintain that the chief feature in the pathogeny of the affection is the angioneurotic element which induces the special spastic œdema. The

significance of various cocci and bacteria which have been discovered in the lesions is doubtful.

It appears almost certain that the affection cannot be regarded as in any sense a specific one, and that the pathology of the erythemata, whether of the roseolar or multiform types, has its explanation in the hypothesis of the presence of various poisons manufactured within the body or introduced from without. This view is rendered almost certain by the fact that erythema multiforme is intimately associated with rheumatism—*i.e.* that it is produced by the action of the toxin of the diplococcus rheumaticus—and that the poisons circulating in the blood in typhus, typhoid, vaccinia, cholera, erysipelas, gonorrhœa, diphtheria, septicæmia, and other affections often produce the typical condition. Moreover, its frequent appearance after the administration of numerous drugs in some individuals, and after the injection of tuberculin and the various sera employed in serum-therapy, proves that it should be regarded as but a symptom of innumerable poisons, and in no strict sense as a specific disease. Schamberg classifies the poisons which cause the various forms of erythema into (*a*) bacterial and protozoal poisons ; (*b*) ptomaines ; (*c*) leucomaines and other metabolic poisons (the cause of the so-called nervous cases really consists in leucomaine-poisoning) ; (*d*) drugs.

Diagnosis. — The varying polymorphous character of the different lesions, coming out in crops and chiefly confined to the backs of the hands and feet, or in the vicinity of joints, or on the face, and the fading away in one region whilst extending in another, is characteristic. If the eruption be viewed as a whole, the features which seem in any written description to be likely to lead to confusion and difficulty render the diagnosis easy and certain, in most cases at a glance.

Measles is easily differentiated from the papular form of the disease by the higher fever, universal distribution of the eruption, the presence of coryza, cough, and conjunctivitis.

Rötheln begins on the forehead or face, and becomes general, but the enlargement of the lymphatic glands behind the sternomastoid is characteristic even when the fever is very slight. The individual spots are less elevated, smaller, and of a brighter red colour than the larger, flat-topped, deep-coloured papules of E. multiforme.

Acute eczema is ushered in with much burning or smarting itch, and mostly without constitutional symptoms ; the individual papules or vesicles are smaller, more pointed, and run together in patches, and the characteristic discharge appears.

Urticaria has a very sudden onset. The wheals appear as large white plaques, which reach their full development in a few minutes, always with severe itching ; they are of brief

duration, and they are not symmetrically distributed. A variety of E. multiforme sometimes described as *E. urticans*, in which patches appear like small red wheals, is liable to be mistaken for urticaria. It is a modified *E. iris*, and is differentiated by its longer duration.

Dermatitis herpetiformis, or *Hydroa*, may closely simulate multiforme; but the itching is intense; pustules and bullæ are often present, which are rare in multiforme; and the eruption tends to become widely diffused, and the tendency to the annular or concentric ringed appearance is absent, though Duhring has seen both these polymorphous skin-affections occurring together.

Syphilis may be mistaken at the beginning of the secondary stage for multiforme, but the local lesion and the secondary lymphatic glandular and throat phenomena usually suffice to establish the diagnosis.

Prognosis.—This is always good as regards the erythema itself, but the primary cause, as rheumatism, septicæmia, or the presence of endocarditis, must be reckoned with in forecasting; and it must be remembered that recurrences are common, and the rule in *E. iris*. The attack is almost certain to disappear inside thirty days, and even at the end of a week nothing may remain but the staining. The prognosis is also good in peliosis rheumatica.

Treatment. — The cause should be removed when it is possible to discover it; thus some drug administered for a chronic ailment may be producing the affection. As the disease has probably a rheumatic origin in the majority of cases, the best routine treatment will be the administration of full doses of purified sodium salicylate, rest in bed, and light diet.

Local applications are seldom required; but in *E. iris*, when there is much itching, evaporating lotions may be useful. In these cases the œdema may be controlled by chloride of calcium internally.

ERYTHEMA NODOSUM

Is usually described as a form or variety of E. multiforme, but it is here referred to separately because of its more definite clinical symptoms and the absence of the usual polymorphous or multiform character of the lesion. Its pathology may be taken as identical with the other forms, though it differs in some etiological features from the affection just described. Thus it is a disease of childhood or of young adult age, appearing rarely in or after middle-life; its relation to rheumatism is by no means obvious, and is denied entirely by many authorities. Girls are much more frequently affected than boys. Recurrences are certainly so very exceptional that they are probably coincidents, and the site of the eruption and its nodular character are almost unvarying.

In these particulars nodosum differs markedly from multiforme; but unquestionably it may occur in its typical site and nodular character during an attack of multiforme, and the writer has witnessed a sparse but characteristic *E. iris* on the hands coming out after the nodes had existed for a couple of weeks on the legs. Stephen Mackenzie insists upon its close association with rheumatism, and has reported cases of endocarditis arising along with it.

Symptoms.—The eruption is practically always confined to the shins, back of the forearm, and elbows, though very occasionally it may be seen on the shoulders, face, and thighs; it has also been reported as affecting mucous membranes. As its name implies, the lesion consists of nodes; several of these, from the size of a split pea to that of a small poached egg, are arranged upon each shin with their long diameter parallel to the tibial edge. The skin is of a bright red or rose colour over each nodosity, the margins of which shade off gradually into healthy tissue. The swellings are very tender and painful. The entire course of the tibia may be the seat of a severe pain before the appearance of the eruption, and smart feverishness and neighbouring joint-pains may precede or accompany the appearance of the nodosities. The eruption is always symmetrical or bilateral; several nodes may coalesce, so that the skin over the front of the tibia may be occupied by a large inflamed, œdematous, tense mass of apparently contused tissue, with slight depressions corresponding to the limits of each node. The colour soon changes to a deep livid or violet as the nodosities soften in the centre, but suppuration or ulceration of the skin never takes place. At a later period they show the familiar variations of colour characteristic of a smart injury, with ecchymosis. At this stage the diagnosis from a series of blows or contusions inflicted over the shin or ulna is impossible except by the history of the case.

The duration of the affection is about a fortnight, but it may be prolonged for a month by the appearance of a fresh crop of nodes.

Diagnosis.—*Syphilitic nodes* appearing during the secondary stage, especially if the syphilitic eruption on the body is of the multiforme type, may cause much confusion in the diagnosis; only the careful recognition of the other manifestations and history of syphilis, especially the condition of the lymphatic glands, will prevent error.

From the slowly forming unsymmetrical nodes of the tertiary period there is little difficulty of differentiation.

Bromide-rashes may present an appearance which cannot be differentiated; indeed, the writer believes that the two conditions are identical, having seen typical E. nodosum produced by large doses of the potash and soda bromine salts.

Prognosis.—The affection invariably ends in complete resolution in a few weeks.

Treatment. — Little interference with the eruption is needed, since it tends to speedy resolution if let alone. The best routine method is to envelop the limbs in a layer of cotton-wool under a moderately firm bandage. Where the tension is high and pain is severe, an evaporating lotion may be applied and the parts elevated. The patient always experiences relief when placed in the horizontal posture.

ERYTHEMA ELEVATUM DIUTINUM

This is a rare form of erythema, described by Crocker and Williams as occurring in children. One case seen by the writer, in conjunction with Dr. J. C. Hall, in a young male adult presented the following characters. There was an eruption of a month's standing on the backs of both hands, consisting of nodules of a red colour which coalesced over the ulnar border; they were firm and sharply defined, and tender on pressure and somewhat painful at night. Several were as large as a small bean; the induration and coloration of the intervening patches of skin presented an unmistakable erythematous character. The larger nodosities or plaques situated over the metacarpal bones of the little fingers and thumbs appeared first, and had remained with little change; but smaller livid pea-sized nodules were of recent growth, and had replaced similar swellings of a bright-red colour which had come out in crops surrounded by an erythematous base, presenting a resemblance to E. iris. There was a similar but smaller form of the nodosities on the dorsum of the feet, and patches on the buttocks. Intermittent albuminuria was well marked, but there was no other sign of deterioration of health. The eruption had followed a severe and acute sore-throat, which was probably of diphtheritic origin. There was a small patch on each knee, about the size of a shilling, which closely resembled psoriasis; this appeared about the same time as the erythematous eruption on the hands and feet.

ERYTHEMA INDURATUM, OR BAZIN'S DISEASE

This is a rare form of skin-affection occurring in young women compelled to spend long periods in the standing posture. The eruption consists of diffuse patches of nodes of the erythematous type, situated generally upon the calves of the legs. These are of an indolent character, somewhat resembling nodosum, of a dull violet colour and flattened on the surface. They are at first deeply seated in the corium, and after they come to the surface they tend to break down and form ulcers with punched-out margins, which are very chronic in their course. The disease has been supposed to

be always of tubercular origin, which is very improbable, as the site of the eruption is always bilateral or symmetrical. Whitfield maintains that there is both a tubercular and non-tubercular type, the latter appearing in older patients and running a more acute course. Alexander and Harttung, according to Norman Walker, have proved the tubercular nature of the lesion in five cases; they maintain that the pathogeny of the tubercular affection is hæmotogenous, and essentially an arterial embolic condition.

FACIAL HEMIATROPHY

This is a very rare affection, characterised by atrophy of all the tissues, including the osseous, upon one side of the face and sometimes of the half of the tongue on the same side.

Etiology.—The affection begins before puberty, after injury or during convalescence from one of the infectious fevers, and often without any apparent cause, and is more common in girls than in boys. The pathology of the affection has been considered as a trophoneurosis affecting the fifth nerve; by Mendel the descending trigeminal root has been found atrophied, and the branches of the nerve the seat of an interstitial neuritis.

Signs and Symptoms.—The first symptom is a whitish parchment-like spot or patch appearing upon some part of the face and presenting characters not unlike sclerodermia, and some authorities regard the condition as the result of sclerodermia. This gradually spreads, and with its increase is noticed a wasting of the subcutaneous fatty tissue and bone, the muscles being but slightly involved, if at all, save by the disappearance of their interstitial fat. The cartilages also waste, the teeth drop out, and the hair of the eyebrows is whitened or shed; the eyeball recedes and appears smaller owing to the absorption of the orbital fat. The withering stops at the middle line, producing a physiognomy which is unmistakable. A few cases are, however, recorded where the affection was bilateral, and some where only a portion of one side of the face was involved. The cutaneous sensibility and movements are unaffected, and the muscles respond normally or more readily to weak electrical stimuli. Occasionally some patches of wasting have been observed on the back and arm of the same side of the body.

Diagnosis.—*Congenital asymmetry* of the face is a very common condition, but, unless when very extreme, it can hardly be confounded with hemiatrophy; there is never associated with it the falling-out or bleaching of the hairs seen in this affection, and the hidebound condition of the skin and its parchment coloration and feeling are absent.

Infantile hemiplegia reveals itself by the loss of muscular movement in the face and other parts, as well as by spasticity and increased reflexes.

Congenital wry-neck is associated with asymmetry of the face, but the shortening of the sternomastoid is obvious.

Poliomyelitis anterior acuta, affecting the facial nucleus, is differentiated by its electrical reactions and the wasting of the muscles.

Paralysis of the sympathetic causes partial ptosis and contraction of the pupil on the affected side, whilst in hemiatrophy this is usually dilated.

Hemihypertrophy of the face is a rare condition characterised by hypertrophy of the osseous tissue and skin, the facial bones, the palate, basilar process, and sphenoid sharing in the general enlargement. It may without care be confused with hemiatrophy, the normal side of the face being mistaken for an atrophied condition.

Prognosis.—The affection is generally progressive, but sometimes its progress is arrested and it may remain stationary for the remainder of life. It does not shorten life, and can only be regarded as serious owing to the deformity which it produces. It is uninfluenced by treatment, according to the experience of all authorities, though the writer in one case believed that arrest followed hypodermics of strychnine; but he lost sight of the case after considerable improvement had set in.

FACIAL PARALYSIS

In order to grasp the different forms of paralysis of the seventh nerve, a brief reference to its anatomy is necessary.

The cortical centre for the facial movements is situated in the lower third of the ascending frontal convolution. The volitional tract from this to the nucleus of the nerve extends downwards, in the knee of the internal capsule, along with the fibres from the leg and arm cortical centres, passing through the cerebral peduncles to the pons. Before reaching the nucleus of the facial nerve, which is situated in the pons, the fibres from the motor cortical centre cross the middle line to the opposite side in the upper part of the pons.

The fibres from the facial-nerve nucleus pass downwards under the floor of the fourth ventricle, and leave the pons in association with the auditory nerve after looping round the nucleus of the sixth nerve. (It is supposed that the root-fibres which ultimately supply the orbicularis palpebrarum and frontalis, before issuing from the pons, are connected directly with the nucleus of the third nerve, and that the hypoglossal nucleus supplies fibres which go to the orbicularis oris.)

The nerve enters the internal auditory meatus with the auditory nerve winding through the Fallopian canal in the temporal bone along the tympanic wall, and, passing out through the stylomastoid foramen, divides ultimately into branches which supply all the muscles of the face, the platysma, the occipito-frontalis, the external ear, the digastric and stylo-hyoid muscles, having previously given off the chorda tympani and a branch to the stapedius muscle.

Pathology and Etiology. — Paralysis of the face may be caused by a lesion in any part of the path above described; thus it may be *supra-nuclear* when the cortical centre or the tract passing from it to the facial nucleus is involved; *nuclear* when the nucleus itself suffers; *infra-nuclear* when the fibres within the pons or outside it during its course to the muscles are involved. In the latter cases the paralysis is often named *peripheral*, in contradistinction to *cerebral* or *central* paralysis caused by cortical or supra-nuclear lesions.

Cerebral, central, or supra-nuclear paralysis is distinguished from nuclear, infra-nuclear, or peripheral palsy by certain definite signs; thus the upper muscles of the face are little affected or escape completely when the lesion is above the nucleus, the patient being able to close his eye and wrinkle the forehead when the angle of the mouth is obviously distorted. Even when he cannot do this voluntarily, both movements may be accomplished under emotional excitement, as supra-nuclear palsy affects voluntary movements more than emotional ones. Owing to the nucleus still exercising its trophic function, the muscles and nerve do not show the reaction of degeneration. Upon the other hand, when the lesion affects the nucleus or the nerve on the distal side of it, all the muscles of the face are paralysed; they speedily degenerate, and the response to faradism is absent, whilst they respond readily to voltaic stimulation. Paralysis of the supra-nuclear type is commonly met with in hemiplegia caused by hæmorrhage or softening, and it is upon the same side as the hemiplegia, because the fibres decussate in the upper part of the pons before entering the nucleus; in this respect the facial muscles are related to the cortex in the same manner as are the leg and arm muscles. When the lesion is confined to the facial centre in the cortex, the mouth-muscles may be paralysed without hemiplegia (*facial monoplegia*).

Crossed paralysis, in which the face is affected upon the side opposite to the hemiplegia, occurs when the lesion is situated in the lower part of the pons affecting the nucleus or the nerve-fibres between it and the point at which the fibres reach the surface. In this case the lesion catches the arm and leg fibres before their decussation in the medulla, and the facial cortical fibres after they have crossed the middle line of the pons, and there will likely be associated with this crossed

paralysis affection of the sixth, fifth, and sometimes of the eighth nerve.

Double or bilateral facial paralysis, or facial diplegia as it is called, is rare; it is caused by bilateral cortical lesions, as syphilitic meningitis, or a lesion at the point of decussation of the facial cortical fibres; but it may be peripheral, as from gummata or fractures of the base of the skull, double otitis media, and bulbar paralysis. More commonly it is the result of diphtheritic neuritis; occasionally it may arise in the ordinary form of so-called 'rheumatic' facial paralysis about to be described, one nerve being affected some time after its fellow on the opposite side of the body.

Nuclear and infra-nuclear paralysis of the facial nerve is often termed 'Bell's paralysis.' It is described as a *peripheral* palsy in contradistinction to the central or supra-nuclear form, as practically the same symptoms result from disease of the nucleus itself as from an affection of the root-fibres or nerve-trunk. The great majority of cases occur from exposure to cold, affecting the nerve-trunk in the Fallopian canal, and it is to these that the term Bell's paralysis is usually applied; hence the affection is also often called 'rheumatic' facial paralysis.

As already seen, the palsy may also result from pontine lesions, as tumours, hæmorrhages, bulbar paralysis, poliomyelitis, &c., from disease or injury at the base of the brain, from otitis media, and temporal-bone disease, syphilis, diphtheria, &c.; in rare instances it has been congenital exclusive of those common cases caused by injuries during birth.

It has been held that Bell's paralysis is the result of an interstitial neuritis, caused by exposure to cold, which involves the nerve in the Fallopian canal, the consequent swelling of the sheath by its pressure destroying its function; but recent authorities regard the affection as the result of degenerative changes in the myelin-sheaths without any interstitial lesion.

Symptoms.—These come on usually within twenty-four hours after severe exposure to cold, especially when a current of cold air has been blowing upon the side of the face, as in sitting opposite an improperly fitted or partially closed window; often the patient awakes with his face paralysed. Pain and stiffness of a rheumatic kind are sometimes complained of over the affected side, and the writer can testify from his personal experience of an attack that this pain may be severe, especially about the region behind the ear. When the paralysis is complete there is loss of power in all the muscles supplied by the nerve on the affected side. The mouth is drawn to the sound side when the features are in repose, and the skin over the affected area is smooth, owing to the absence of wrinkles and furrows; this in the aged is very characteristic. The loss of power is evident on smiling, laughing, or crying, and

on any attempt to whistle, puff out the cheeks, close the eyelids, or throw the skin over the forehead into wrinkles. Thus the attempt to wrinkle the brow succeeds on the sound side, but the furrows stop in the middle line; the eyelids cannot be brought together on the affected side, but the eyeball turns upwards under the upper lid, exposing the sclerotic only, and the patient is unable to blow out a lighted taper.

Labials are imperfectly articulated, so that speech may be somewhat altered; but there is no weakness of the tongue, and any apparent deviation from the middle line when the organ is protruded results from the alteration of the oral aperture by the paralysed angle of the mouth. The food collects between the teeth and cheek in eating; and in drinking fluids may run out of the mouth at the paralysed angle. In forced breathing through the nose the nostril on the affected side does not expand, and the tip of the nose may incline to the sound side. The asymmetry is rendered very evident by getting the patient to attempt to expose his upper incisors whilst he strives to firmly close both his eyes. The weakness of the platysma may be observed when the patient attempts to depress his lower lip.

Reflex movements are lost; and since the eyelids do not guard the conjunctiva from foreign bodies, conjunctivitis and watering of the eye are common. There is no evidence that the palate suffers in the paralysis, though the uvula may deviate—a common condition in the normal subject. There may be diminished sense of smell in the affected nostril owing to impaired power of dilatation.

Taste is deficient or lost in the anterior two-thirds of the tongue on the affected side through the chorda tympani being involved. The presence of this symptom shows that the nerve is affected at a point between the origin of the chorda and the geniculate ganglion in the bony canal, and according to Gowers it is lost in half the cases resulting from cold. It is not lost when the lesion involves the fibres in the pons, or the root-fibres, or the trunk of the nerve outside the skull.

Hearing may be more acute than normal through the paralysis of the stapedius, but it is deficient when the auditory is involved with the facial in the petrous portion of the temporal. Sensation is normal or but altered to a trifling extent when pain is present. The electrical reaction may present the changes typical of degeneration; thus, in a severe case, in about twelve days the excitability of the *nerve* by the *faradic* current is lost, and the excitability of the *muscles* by the same current is also lost. The excitability of the *nerve* by the *galvanic* current is lost, whilst the excitability of the *muscles* by the *galvanic* current is increased or modified. In mild cases the irritability of the nerve may be only diminished; in all cases ending in recovery the irritability of nerve and

muscles returns slowly, but usually not till some voluntary power in the muscles has become obvious. Wasting of the muscles is not marked unless in long-standing cases.

The course of the affection varies with the severity of the nerve-lesion; in mild cases all paralysis may disappear inside a fortnight, but the usual duration approaches nearer to two or three months, by which time, if marked improvement has not set in, ultimate recovery usually will be found incomplete.

Over-action or muscular *contracture* may supervene in severe cases, and this may be so marked as to lead to the error of mistaking the weakened side of the face for the sound side. In aged patients this secondary change may lead to restoration of the symmetry of the lower part of the face in repose, but in young subjects it may aggravate the deformity. As the muscles shorten, the mouth is drawn towards the paralysed side, the lost naso-labial fold returns in an exaggerated form, and the movements of the muscles are exaggerated on the affected side in laughing or speaking; in smiling, the eyelids of the affected side may almost close from over-action of the orbicularis palpebrarum. If, however, the patient be directed to bring any group of muscles into strong action, as in forcibly closing the eyelids and raising the upper lip, the preponderating power of the sound side of the face will become evident.

Ultimately the affected muscles, especially the zygomatici, may become the seat of spasmodic twitchings.

Diagnosis.—The chief points in the diagnosis between central and peripheral paralysis have been already mentioned upon page 415; the presence of the reaction of degeneration and the obvious involvement of the O. palpebrarum demonstrate that the lesion is nuclear or peripheral. The position of the tongue is important, being usually deviated towards the affected side in central paralysis. The localisation of pontine lesions by the presence of crossed paralysis and accompanying paralysis of the fifth, sixth, and eighth has been already mentioned, but a lesion of the pons is certain if all the parts of the nerve are affected save those going to the O. oris; when this facial muscle only is affected the tongue will be found also to suffer, the lesion being close to the nucleus of the hypoglossal. Lesions about the base of the brain, as meningitis, gummata, and injury, may be inferred when the auditory is paralysed in the absence of ear-disease.

When taste is unaffected, the lesion is in the lower part of the bony canal or outside the stylomastoid foramen.

The difficulty in determining the side involved when over-action has supervened has already been dealt with.

Prognosis.—In pontine lesions recovery is not to be expected; when the paralysis is due to ear-disease recovery is rare, and the same result usually follows if a sudden hæmorrhage

into the nerve has been the cause. Syphilitic lesions about the base of the brain may yield to active treatment, which will cause the paralysis to disappear. Injury to the nerve during delivery generally ends in recovery, and a similar result may be expected in diphtheritic cases. The important point in prognosis is the future condition of the so-called rheumatic cases or those resulting from exposure; and though the forecast is usually favourable in these, a percentage of the cases end in permanent deformity.

The electrical reaction affords the only means of forecasting the ultimate result. Thus, if by the tenth or twelfth day the faradic irritability of the nerve remains good, the paralysis will probably disappear in three or four weeks. If the irritability be completely lost at the end of a fortnight, six months' duration of the paralysis may be predicted. Recovery may also ensue even in cases where faradic irritability remains absent for two or three months; but in these cases over-action of the affected muscles is liable to occur and become permanent, though ultimately the over-action may become less noticeable. When only a partial reaction of degeneration is obtainable within a fortnight after the onset, the case may be regarded as likely to clear up within eight or ten weeks.

Treatment.—In the rheumatic cases the best routine plan is blistering behind the ear, or leeching when pain is prominent, rest in bed, and the application of a large pad of absorbent wool to the affected side of the head and face.

Salicylates may be given when any increase of temperature is present, but full doses of iodide will probably give best results in most cases. Electricity, though possessing no influence over the progress of nerve-degeneration, is of undoubted value in preserving the tone and nutrition of the muscles till the nerve recovers its function. Galvanism should therefore be resorted to early (within a fortnight). Some authorities, including Risien Russell, consider that faradism is liable to cause over-action ultimately if persevered in, but this result is noticeable in cases where electricity has never been employed (Mayer). In all cases, however, a weak current only should be employed, and electrical treatment should be given up as soon as over-action appears. Facial massage and hypodermics of strychnine may be employed in long-standing cases.

Where syphilis is suspected, and where middle-ear disease is present, the appropriate agents should be pushed. Surgical measures, as suturing the ends of the nerve divided in mastoid operations and other injuries, have been successful in some cases.

FACIAL SPASM

This, also known as Convulsive Tic or Mimic Tic, is a painless clonic or tonic spasm affecting one or more groups of the muscles supplied by the facial nerve, generally on one side of the face.

Etiology.—A neurotic family history is the rule, and women are more frequently affected than men, the disease usually appearing in its typical form about the age of fifty years or at the climacteric period. Severe mental emotion, shock, prolonged worry, and grief are often traceable as the cause, and many cases appear to arise in some reflex way from peripheral irritation about the orbit and teeth, or from exposure of the skin of the face, head, or neck to severe cold or heat. In these cases neuralgia may be severe at the time of the irritation, but the spasm remains after all pain has disappeared. (The spasm which often is associated with severe neuralgia of the fifth nerve does not come within the category of simple facial spasm.) Uterine and visceral irritations are supposed to be the cause in some cases; but it is rarely associated with pregnancy. Sometimes the condition appears to be hereditary, as in those cases which occur in childhood, and in which there is often a mental condition in which inhibitory influences are weakened, as shown by the explosive utterance of certain words or the production of strange noises.

Paralysis of the facial nerve ending in contracture or over-action of the weakened muscles, which become the seat of chronic spasmodic twitchings, and irritation of the facial motor cortex and the facial nucleus by some structural lesion, are accountable for a number of cases.

The continually repeated performance of some movement of a group of facial muscles may induce spasm, though such cases probably should be included under 'habit' spasm, as the winking and grimaces of neurotic children. The action of the orbicularis in protecting the eye from light in photophobia causes the variety of facial spasm known as Blepharospasm, which affects the eyelids; and a somewhat similar condition may be seen in watchmakers from the use of the lens employed in their work, and more rarely in those individuals using a single eyeglass for myopia.

Pathology.—Exclusive of the very small number of cases depending upon obvious or gross organic lesions in the cortex and the pons, the great bulk of the cases must be regarded as functional or idiopathic, and about the pathology of these there is much difficulty. The probable explanation of the spasms is an instable condition of the cells in either the cortical centre or in the facial nucleus in the pons, which, according to Gowers, produces a discharging lesion. When the cause

is some pressure upon the trunk of the nerve it is probable that this has affected the nucleus. In a small percentage of cases the cause may be a reflex one.

Symptoms.—The spasms are usually confined to one side of the face; occasionally they are bilateral. They are chiefly clonic in character, consisting of a series of rapidly recurring contractions of the muscles of one or more groups supplied by the facial nerve, which may produce grimaces of various types. These clonic spasms may eventuate in a true tonus of several seconds' duration, or the tonic and clonic contractions may alternate; or the clonic contractions may be so rapid as to pass unobserved, when the attack may present all the features of a true tonic spasm. All the facial muscles on one side may be affected, but sometimes the spasm remains confined to the zygomatici, or to the elevator of the lip or of the alæ nasi; or it may originate in these and rapidly spread to all the muscles supplied by one facial nerve, except the digastric or stylohyoid. Rarely is the spasm confined to the ear-muscles or the orbicularis oris or the frontalis.

Blepharospasm, as already mentioned, is the name given to the type which confines itself to the orbicularis palpebrarum; it is usually bilateral, caused by photophobia, and is of a *tonic* character. The eyes may remain closed for several hours or many days, even during darkness, but more commonly each attack only lasts for some minutes. There is a *clonic* variety of this type, known as nictitating, or winking spasm, which is usually a true habit-spasm or of hysterical origin.

Facial spasm is increased by emotional states, and by all movements of the muscles, as in laughing, reading aloud, speaking, or chewing; it is intensified by a current of cold air or by bright light or by palpating the muscles or skin, and it is lessened by repose. The attack of spasm may last for several seconds or minutes, and the intervals may extend from several minutes to hours. The attack is painless and there is no loss of power in the affected muscles, except in those cases depending upon organic lesions, and the electrical reaction is normal.

In severe blepharospasm a continuous noise has been experienced by the patient, which has been supposed to be the result of spasm of the stapedius muscle, but this very rarely occurs as the result of spasm confined to the muscle. Occasionally spasm of the palate-muscles has been noticed, and rarely the contractions in the facial muscles have been observed to extend to those of the tongue, neck, and jaws.

True tonic spasm of the facial muscles has been observed to follow an attack of Bell's paralysis or tetanus. It has been seen to follow exposure to cold which did not cause palsy, but usually it is associated with hysteria.

The course of facial spasm is very varying. It may appear suddenly, but usually it begins gradually in some slight twitching movements which slowly increase in intensity and spread to neighbouring muscles, often pausing for long intervals or entirely disappearing for a time, to recur with increased severity. It may extend from the muscles of one side and involve the corresponding ones on the opposite side. Usually its duration is chronic: it is prone to remain for years or during life.

Diagnosis.—*Hysteria* may produce a tonic spasm of the O. palpebrarum, but the other phenomena of hysteria are always in evidence; the quivering which is common in the facial muscles in hysterical patients presents distinct emotional characteristics which can scarcely be mistaken.

Chorea may simulate facial spasm, but a close examination of the limbs will always demonstrate the characteristic choreiform movements somewhere. The diagnosis of cases depending upon an organic lesion is made by detecting actual loss of power in the muscles and evidence of implication of other cranial nerves, or of hemiplegia. It is always necessary to differentiate idiopathic spasm from that which comes on after paralysis of the facial nerve; in this latter case there will be contracture or over-action, with real loss of power in the muscles which have become the seat of spasm.

When all the muscles upon one side of the face are affected the spasm probably originates in an irritable condition of the facial nucleus; but when only the zygomatici are involved the fault probably lies in a similar condition in the cells of the cortical centre.

Prognosis.—This is generally unfavourable in all cases which have persisted beyond a few months' duration, and when of long standing it may be predicted to be a permanent or lifelong annoyance in the great bulk of cases. Recent cases of reflex origin may be cured by the *prompt* removal of the exciting reflex cause, as those resulting from nasal, eye, and teeth irritation. Spontaneous recovery is rare, but remissions are often noted. There is only danger to life in those rare cases originating in a progressive organic malady.

Treatment.—This, as a rule, is most unsatisfactory, save in the reflex cases where the removal of the cause may be effective. Blepharospasm is more likely to disappear upon treatment of the eye-mischief than the other forms of facial spasm.

Sedative drugs, as large doses of bromides, Indian hemp, gelsemium, hyoscine, belladonna, morphia, &c., only give temporary relief, and their prolonged administration is objectionable.

Local measures sometimes afford considerable relief for a short time, and foremost amongst them is blistering, or the

cautery applied behind the ear. Electricity in every form has been tried, but only a weak galvanic current is admissible, applied by the stabile or continuous method. Mayer recommends resection of the supra-orbital nerve where this is sensitive to touch, and where pressure over it will inhibit the spasm. Stretching of the facial nerve will stop the spasm for many weeks, and in one or two cases cure has been reported ; but the rule is that after the paralysis which follows the operation has disappeared the spasm returns.

FARCY. (See GLANDERS.)

FAVUS

This is a contagious affection of the skin caused by the presence of the Achorion Schönleinii fungus.

The parasite usually infests the scalp, but it may attack any portion of the skin or the nails, and it has been found in a fatal case on the gastro-intestinal mucous membrane.

Etiology.—The disease is common in mice ; from them it is transferred to the cat, which is supposed to be the usual source of human infection. It is common in Scotland, though rare in England and seldom met with in Ireland, but very prevalent in Russia and Poland.

Symptoms and Signs. — The affection begins with a slight superficial patchy irritation of the skin of the scalp, which soon produces a small cup-shaped primrose-coloured disc, which grows in the epidermis, out of which it gradually emerges. The centre of the cup or *scutulum*, as it is called, is perforated by a hair, and as the cups increase in size they lose their circular outline by compression of adjoining cups, and thus become hexagonal in outline, resembling a piece of honeycomb, which characteristic resemblance gives the name to the disease. After a time the scutula coalesce into ragged crumbling crusts, not unlike pieces of yellow cheese with worm-eaten markings, in which the individual cups can only be recognised about the circumferential zone. Eventually a large portion or the entire scalp may be covered by these crusts, underneath which atrophic scarring occurs, with atrophy of the hair-bulbs, often producing permanent baldness.

The crusts have a characteristic mouse-like odour ; pediculi are often present, as the disease usually appears in the scalps of filthy and neglected children, and much itching and pustulation may thus be produced. The disease, if untreated, runs a very chronic course, and flourishes till every hair-

follicle is destroyed. Norman Walker reports a case in which the disease had lasted for fifty years.

Diagnosis.—This is easy, as the established affection can hardly be mistaken for any other condition. Once the writer had a momentary difficulty in recognising the disease owing to the prevalence of an extensive impetigo, but a perfectly formed sulphur-coloured cup on the chest, measuring one-third of an inch in diameter, was conclusive ; the odour of the crusts was also diagnostic.

The microscopic examination leaves no room for doubt in any case ; a fragment of a cup moistened with liquor potassæ shows the long tubes of characteristic mycelium forming a felt-work, in the meshes of which the shorter spores may be seen. These spores maintain their vitality for years, and they undergo such changes in culture-experiments as have led to the opinion that several distinct fungi may cause favus.

Treatment.—This is usually most tedious and disappointing when the disease has been neglected for a long time before coming under observation. In early cases it is more satisfactory. All crusts must be removed by poulticing or oiling, or by the application of compresses of saturated boric solution under oiled silk. Every hair in the affected region must be epilated by forceps or other methods, and then the remedies suitable for ordinary ringworm-treatment must be applied till the destruction of the fungus is accomplished. Blistering is most useful, and alone may effect a cure where the affected region is limited. Walker speaks highly of the method of causing the shedding of the hair by exposure to the X-rays, instead of the tedious process by epilation. Recurrences are common, and the treatment must not be discontinued till after every trace of the disease has been removed, which usually means a matter of many months.

Baldness is not to be expected when the treatment is commenced early. When the nails are infected avulsion may be necessary.

FEBRICULA

This term is applied to a febrile condition of a few days' duration, which is supposed to be independent of invasion by any recognised bacilli or organised virus. When the fever disappears at the end of twenty-four hours the case is styled as one of ephemeral fever.

The term is convenient : it implies ignorance of the cause of the increased temperature, and permits the classifying together of a number of different conditions, having nothing in common but some disarrangement of the heat-centres.

Etiology and Pathology.—The largest number of cases are supplied by indiscretions in diet which permit of the

introduction of toxic substances in the food, or which cause such digestive trouble as leads to the formation of some poison in the intestine. Many of the temporary fevers of childhood find their origin in such digestive disturbance, and dyspepsia in the adult is often accompanied by fever of brief duration ; hence the term 'gastric fever,' which is, however, wrongly applied to typhoid.

The abortive attacks of fever which are often witnessed in epidemics of typhoid and other members of the exanthemata are usually grouped under Febricula, though they are probably caused by the toxins produced by the specific bacteria before these have been destroyed by the phagocytes which cut short the affection.

Febricula of short duration appears to be producible by the interference with the thermic mechanism which results from exposure to cold or to solar heat. Whether the increase of temperature in such cases is the result of increased thermogenesis or diminished thermolysis, or the outcome of a disarrangement of the thermotaxic or regulating mechanism, is not capable of demonstration.

Prolonged severe muscular exertion sometimes causes smart febricula which is probably the result of over-production of heat in the muscles, though normally the muscular source of heat is independent of contraction of their fibres.

Sewer-gases, when inhaled by persons unaccustomed to these exhalations, often produce brief pyrexia so suddenly following their introduction as to lead to the conclusion that the phenomenon is independent of bacterial infection.

Exclusive of the above-mentioned causes of febricula, in a large proportion of the cases no clue to the origin of the fever is forthcoming, Cases of tonsillar source should not be included under Febricula, as these, though of short duration, are almost certainly of bacterial origin.

Symptoms. — The symptoms are those common to all fevers in their early stage ; the invasion is abrupt, with smart malaise, head and backache, the temperature rapidly rising to 101° or in children to 104°. The skin is dry, the urine scanty and high-coloured ; herpes on the lips is commonly present, especially in cases the result of exposure to chills. The crisis occurs at the end of the first, second, or third day : occasionally it may be later ; it sets in abruptly, usually with profuse perspiration.

Diagnosis. — This can only be made after the expiration of the period which excludes the appearance of the ordinary exanthematous rashes ; hence febricula is usually only demonstrable after the advent of the crisis. Irritation of the urinary bladder is often the source of smart transient fever in cases of obstructed flow or cystitis, and it would be well that this should be borne in mind in investigating the causes of fever in elderly male patients.

Treatment. — Little is necessary beyond rest in bed and milk-diet ; a mild saline purge is beneficial in all cases. It is advisable to avoid the use of the newer antipyretic drugs, which may cause rashes complicating the diagnosis. The old-fashioned diaphoretic combination of sweet spirit of nitre with Mindererus spirit meets all requirements.

FEVER

Each specific fever will be found described under its own name.

FILARIASIS.

(See under CHYLURIA, p. 218.)

FLATULENCE.

(See under DYSPEPSIA, p. 331.)

FRAMBŒSIA.

(See under YAWS.)

FRECKLES

This condition, also known as Lentigo and Ephelides, is characterised by the presence of numerous circumscribed small maculæ, produced by the deposition of pigment (melanin) in the cells of the Malpighian layer of the skin in parts exposed to the sun's rays.

The brownish spots, which resemble somewhat the markings on the egg of a turkey, commonly appear about the end of the first decade upon the sides of the nose and cheeks and the back of the hands, especially in children with red hair. They may fade or entirely disappear in winter, to return after a brief exposure to strong sunlight in the summer. They are of irregular shapes, and may vary from the size of a millet-seed to that of a pea. Though they tend to disappear after middle-life and their presence is indicative of no departure from health, they sometimes cause considerable disfigurement. Occasionally they appear on covered parts, when according to Unna, such cold freckles are to be regarded as pigmented nævi; but apparently identical marks may be observed on the thighs and trunk of patients affected with rheumatoid arthritis and many other affections.

Treatment is unsatisfactory except when the spots are few and large, when the local remedies useful in chloasma may be applied. A weak corrosive-sublimate solution in almond emulsion, 1 gr. to each ounce, may be tried, or the

individual spots may be touched with pure carbolic acid or a paste made with salicylic acid and glycerin.

Protection of the face from direct sunlight when possible always minimises the disfigurement.

FRIEDREICH'S ATAXIA

This, also called Hereditary Ataxia, is an hereditary affection of the spinal cord, characterised by symptoms and signs in many respects resembling ordinary locomotor ataxia.

Etiology.—The disease, though called 'hereditary,' is but seldom transmitted by direct heredity. It is a family disease, several members of the family in the same generation being usually affected. The congenital influence which produces it has nothing to do with syphilis, but a neuropathic history showing consanguinity in the parents is obtainable in several cases. Epilepsy and insanity have been noticed in members of the family who escaped the ataxia. Males are affected more frequently than females in the proportion of three to two, though in some families the boys suffer most, whilst in others only the girls are affected.

The disease shows itself most frequently about the sixth to the twelfth year, though it may appear as early as the second; it is uncommon for it to appear after twenty.

Morbid Anatomy and Pathology. — The chief lesion is a combined sclerosis of the lateral and posterior columns of the cord, the latter being usually more intensely sclerosed than in tabes. In the lateral columns the peripheral portion and the pyramidal tracts are always involved, the direct cerebellar tract showing degeneration, with which is usually associated atrophy of the posterior vesicular column of Clarke affecting both fibres and cells.

The anterior columns are sometimes degenerated close to the anterior median fissure through the involvement of the anterior pyramidal tract. The posterior nerve-roots are more or less affected, but the nerve-trunks in the limbs escape, and only trifling changes have been noticed in the cells of the anterior cornua in a few cases.

The spinal cord is somewhat diminished in size throughout its extent, and sometimes there is atrophy of the cerebellum, pons, and medulla.

The pathology of the condition probably depends upon arrested development or some congenital defect diminishing the vitality of the structures, which ultimately degenerate, though Déjérine regards the affection as resulting from an increase of the elements of the interstitial tissue of a non-inflammatory nature.

Symptoms. — The ataxia first shows itself in the legs by the unsteadiness in gait, the patient swaying from side to side like a drunken person, the feet being kept wide apart to prevent falling. He can only stand with his legs widely separated; closure of the eyes may increase, but often it has no effect upon his unsteadiness. The loss of coördination at a later stage, when the trunk has become affected, may show itself by the patient's inability to sit without swaying. The arms soon become affected, and jerky irregular movements are apparent on the exercise of any volitional effort—these closely resemble chorea—and the head participates in the motor irregularity, or coarse tremors or fibrillary twitchings may develop which are beyond control of the will. The knee-jerks are usually absent when the symptoms first attract attention, but usually there is no loss of the light-reflex, and no marked loss of sensation anywhere save in the last stages of the disease.

Lightning-pains, joint-changes, optic atrophy, squint, bladder-trouble, and visceral crises are not met with save in very rare cases.

Nystagmus is nearly always present, and the speech is very generally seriously impaired, some words being slowly articulated whilst others are clipped; but the intellect and memory remain normal in most cases.

There is often deformity of the feet, the great toe being extended whilst its distal joint is flexed; and after paralysis of the legs, which usually shows itself at a late stage, there are frequently talipes equinus or equino-varus and spinal deformity. Babinski's sign is usually present.

The duration of the affection may extend to thirty or forty years; but its rate of progress is most uneven, some children becoming soon unable to walk, whilst others continue to move about without great difficulty for years.

Diagnosis.—The presence of the cerebellar type of ataxia in more than one member of a family coming on in early life, with nystagmus and disorder of speech, associated with feet-deformity and loss of knee-jerk, is characteristic.

Disseminated cerebro-spinal sclerosis may be eliminated by the early appearance of ataxia, the loss of knee-jerks, the normal state of the discs, and the absence of spasticity, with increase of the deep reflexes. The wide range of the ataxic jerky movements is not likely to be confused with the finer intention-tremor of disseminated sclerosis, and the staccato syllabic scanning speech differs materially from the speech-disorder of Friedreich's ataxia, which in advanced cases may become, as described by F. T. Roberts, an unintelligible jumble.

Cerebellar tumour may produce an ataxic gait, but the arms are free, and there is usually optic neuritis, severe headache, or vomiting.

In Marie's cerebellar variety of Friedreich's ataxia there are more marked cerebellar symptoms, but the disease sets in after the twentieth year, and the reflexes are normal or exaggerated, with spasticity of the legs, and there are eye-paralyses and optic atrophy ; the deformity of the feet and spine is absent. Direct hereditary transmission is the rule, and the chief lesion is cerebellar atrophy.

Locomotor ataxia is differentiated by the age of the patient, the history of the case, and by the presence of the signs already referred to, which do not appear in hereditary ataxia —viz. the Argyll-Robertson pupil, optic atrophy and eye-paralysis, lightning-pains and visceral crises, trophic joints and perforating ulcer of the foot, involvement of sphincters, and cutaneous anæsthesia.

Hereditary chorea comes on after the age of thirty ; the twitchings begin in the face and afterwards involve the arms, and there is progressive mental deterioration. Ordinary chorea can scarcely be mistaken for hereditary ataxia.

Inherited syphilis may produce ataxic symptoms, which might be mistaken for hereditary ataxia, but they usually come on more acutely and involve the eye-muscles and the optic disc. The Argyll-Robertson pupil will be present, and a spastic condition of the limbs is usually the result of any paralysis. The disease produced by syphilis runs a more rapid course, and epileptic convulsions are common.

Prognosis.—This is always grave. Recovery never occurs, but in some cases the progress of the malady is long delayed, and the patients usually die from other causes.

Treatment.—No drug possesses any influence over the degenerative process in the cord. Much may, however, be accomplished by training the patient by Frenkel's method to re-learn by constant practice the performance of the co-ördinated movements which the progress of the disease has abolished.

GALL-BLADDER, CANCER OF

This is associated with gall-stones in about 95 per cent. of the cases in which it has been found ; occasionally it is secondary to hepatic or pancreatic cancer. It is much more common in women, and often exists as a primary cancer in old subjects.

Symptoms.—The history and early symptoms are those of gall-stones, and deep jaundice is present in the majority of cases ; it is always present where the growth extends to the ducts. When the lymphatic glands at the transverse fissure of the liver become invaded ascites supervenes. There are usually severe paroxysms of pain, liable to be followed by

attacks of fever of the malarial type, with distressing vomiting and occasional hæmatemesis.

In the great majority of cases the gall-bladder may be felt below the costal arch as an uneven, very hard tumour, which may not be very sensitive to manipulation, but which is usually the seat of more or less continuous pain between the paroxysms. The tendency of the tumour is to soon become fixed, so that when seen at a late stage it may not descend on inspiration.

The chief and important feature of the disease is the progressive emaciation and loss of strength. The jaundice is liable to be associated with hæmorrhages which hasten the progress of both these symptoms. Often the diagnosis can only be cleared up by an exploratory incision. Removal of the gall-bladder has been successful in a few cases where the growth was of the epithelial variety ; but, as a rule, excision of the tumour will be found impossible.

Cancer of the bile-ducts is always associated with intense jaundice ; it is practically always a sequel of gall-stones. The gall-bladder swells into a considerable tumour before the appearance of jaundice when the growth affects the cystic duct. When the common duct is the seat of the cancer, jaundice comes on before the gall-bladder enlarges ; but when the growth affects the hepatic duct, though the jaundice comes on early, there is no tumour of the gall-bladder. It is very seldom that the growth itself forms a palpable tumour ; but sometimes when the gall-bladder has been contracted by previous disease, Robson points out that the dilated common duct may be felt through the abdominal parietes, distended like a gall-bladder. The diagnosis is often impossible without an exploratory incision, and removal is generally out of the question.

GALL-BLADDER AND BILE-DUCTS, INFLAMMATION OF

Acute catarrh of the gall-bladder, or catarrhal cholecystitis, is probably always a part of the catarrhal inflammation which affects the bile-passages in jaundice of obstructive origin, whether caused by gall-stones or other obstructions.

Chronic catarrhal cholecystitis may, however, be met with independent of jaundice and gall-stones. The symptoms are those which might be produced by mild attacks of hepatic colic, being caused by the passage of thickened mucus through the cystic duct, and there is little, if any, enlargement or tenderness over the gall-bladder.

The condition is probably the result of causes which interfere with the periodical evacuation of the organ, as in ptosis of

the liver, tight-lacing, indolent habits, and chronic constipation, with scybalæ in the right bend of the colon. It may be the result of gall-stones which have already passed into the intestine, or which have been long retained in the gall-bladder; but it is a well-recognised clinical fact that this receptacle may be filled with concretions for years without producing symptoms of any kind.

There may be great difficulty in the diagnosis in excluding gall-stones, but the symptoms will probably yield to mild purgation and improved hygiene. Robson points out that drainage of the gall-bladder has been curative in those cases where operation has been undertaken under the idea that gall-stones were present.

Suppurative catarrh, or suppurative cholecystitis, as its name implies, terminates in empyema of the gall-bladder.

Etiology and Pathology.—It is often associated with gall-stones, but may arise independently of them, as in typhoid fever, pneumonia, cancer, &c.

The condition is caused by infection either with the *B. coli communis*, *pneumococcus*, *B. influenzæ*, *staphylococcus*, *streptococcus*, or *B. typhosus*, though cases are occasionally met with where the pus is quite sterile. A strange point in infection of the biliary passages and of the gall-bladder is that the bacilli from the intestine are not likely to enter the ducts or gall-bladder till these are blocked by calculi, the outward flow of bile towards the intestine being in the normal condition protective. This fact has been demonstrated by tying the common duct, when the B. coli will appear in the gall-bladder, being absorbed from the intestine in a virulent form.

Symptoms.—These usually come on with marked paroxysmal pain in the right hypochondrium, or even in the region of the appendix, soon followed by some fever with vomiting, and subsequently by chills. In the non-calculous cases the pain is more or less continuous, and there is usually no jaundice. In both types there is marked tenderness over the region of the gall-bladder, and the formation of a painful tumour. When suppurative cholangitis complicates the case, as often happens, the symptoms are more severe: the liver enlarges, fever is high and irregular; deep jaundice is always present whether there be calculi or not, and the prostration is usually extreme.

Intestinal symptoms are usually present, as distension, obstinate constipation amounting to obstruction, and vomiting which may be uncontrollable. If prompt surgical measures be not undertaken, perforation of the gall-bladder and a fatal peritonitis may result, but sometimes the patient may escape with a fistulous opening into the bowel or through the abdominal wall.

Diagnosis.—This may be most difficult where there is no history of gall-stones or jaundice, but the condition may be strongly suspected if the attack comes on after pneumonia or in the late stages of typhoid fever. In a case recently seen by the writer, where surgical aid was rejected, a rapidly fatal issue suddenly followed perforation and a large gall-stone could be felt loose in the lower part of the abdomen; there was no jaundice, but a well-marked history of gall-stones. Some cases closely simulate appendicitis or acute intestinal obstruction; the local tenderness very often does not correspond to the situation of the gall-bladder.

Treatment.—This consists, after an exploratory incision has cleared up the diagnosis, in the free evacuation of the contents of the distended and inflamed gall-bladder, the removal of the obstruction when possible, and the establishment of free drainage.

Acute phlegmonous or *gangrenous cholecystitis* is a rare disease, usually associated with gall-stones. Its pathology is similar to the last described variety, the infective agent being probably an unusually virulent form of the B. coli. Robson points out its pathological resemblance to gangrenous or sloughing appendicitis, with which, however, it is very rarely associated clinically.

The symptoms resemble those met with in the last-mentioned variety, only they set in with great intensity and suddenness, with pain and inflammation in the hepatic region, which soon runs into a severe general peritonitis accompanied by extreme and alarming prostration, vomiting, abdominal obstruction, and rapidity of pulse, the condition resembling that following perforation of a gastric or duodenal ulcer or a perforative appendicitis.

Death may result in two or three days, or sooner, unless surgical procedures be undertaken promptly. These consist in abdominal incision and the evacuation of the contents of the gall-bladder, and its complete removal if gangrene has set in.

CHOLANGITIS

Cholecystitis, as already stated, is often associated with *Cholangitis* or inflammation of the bile-ducts. The *acute catarrhal* form of this, which is independent of the presence of gall-stones, will be described under 'Jaundice.'

Chronic catarrhal cholangitis is usually a part of the clinical picture caused by gall-stones.

Etiology.—It is often termed *Infective cholangitis* because the presence of a stone in the common duct, by preventing the downward flow of bile, permits of the infective organisms already mentioned gaining an entrance. Osler points out that when the obstruction is *complete* in the common duct, the bile-

ducts and the gall-bladder will be found dilated with colourless *sterile* mucus, and there is intense jaundice, usually unaccompanied by fever.

When the obstruction is *incomplete*, whether owing to gall-stones or growths, the introduction of infective agents changes the clinical picture, and fever of the malarial type, first described by Charcot as *hepatic intermittent fever*, becomes pronounced.

Symptoms.—These are rigors, pyrexia, and profuse sweating, which soon follow the severe pain caused by the entrance of the calculus into the common duct. Attacks recur at irregular intervals of weeks or months, in which there may be no fever and in which the jaundice may partially or entirely disappear and the stools become coloured. The gall-bladder is usually not markedly enlarged, but it is generally tender, and ultimately the liver swells and becomes painful after each attack, which is followed by severe jaundice. This condition may last for many months, after which recovery may occur if the gall-stone passes into the intestine. The explanation of the symptoms in some cases is found in the shape and size of the calculus (or calculi), which acts as a ball-valve in the dilated extremity of the duct near its entrance into the duodenum, known as the ampulla of Vater. At any period the symptoms may become grave by increase in the virulence of the bacteria, when the case passes into the following type—viz. :

Suppurative Cholangitis.—This may occur, as just mentioned during the course of a chronic infective cholangitis, or it may suddenly supervene in gall-stone cases, in typhoid fever, cancer, hydatids, suppurative portal phlebitis, or blocking of the duct by an ascaris or other foreign body.

Symptoms.—As the condition is usually associated with gall-stones, there is severe paroxysmal pain followed by jaundice. The fever is severe and of the true pyæmic type, associated with rapid prostration and emaciation, and leucocytosis is well marked. The gall-bladder usually contains pus, and is generally somewhat distended. Rapid enlargement and pain mark the condition of the liver; the enlargement is smooth and uniform at first, but as the suppurative cholangitis involves the biliary passages, abscesses of the liver may destroy the symmetry of the swollen organ.

In rare cases pain may be almost absent; this is more likely to happen in non-calculous cases. Ulcerative endocarditis may be present.

Death is certain unless prompt evacuation of the purulent contents be decided upon, and when the suppuration has become general there is very slight prospect of a successful issue even with all the resources of surgery; but thorough drainage after cholecystotomy will always afford amelioration of the suffering.

GALL-STONES, or CHOLELITHIASIS

These are concretions which are formed in the biliary passages, nearly always in the gall-bladder.

Pathology.—Biliary calculi consist mainly of cholesterin, which exists in a crystalline form laid down in concentric layers and cemented together by lime-salts (bilirubin-calcium). The origin of cholesterin is regarded by Naunym to be the mucous membrane lining the gall-bladder and biliary ducts, and that it is not merely separated from the blood by the liver, but is a true secretion of all mucous membranes, especially when these are in an irritated or inflamed condition. The source of the lime-salts is probably the same, and the authority mentioned believes that the *bacillus coli* and the *bacillus typhosus* play an important part in the irritation which leads to the formation of calculi, which is therefore the result not of hepatic disturbance, but of catarrhal conditions of the mucosa of the gall-bladder associated with stagnation or retarded flow of the bile. The view is steadily gaining ground that micro-organisms play the chief *rôle* in the production of biliary calculi since Fournier and Gilbert produced the condition experimentally in dogs, and this view has pushed aside the old theory that stagnation of bile was the chief factor. Bile-pigment, bile-salts, mucus, and epithelial cells are also present in most cases, and even micro-organisms have been detected. The nucleus is usually a particle of bile-pigment or bilirubin-calcium, or bacilli, or a cluster of epithelial cells. The calculi vary in size from a grain of sand up to stones as large as a bantam's egg; the former is often called bile-sand. When few and large they are usually faceted from contact with each other; the surface is smooth, but it may be mammillated; the colour is a greenish-brown usually, but it may vary from grey to dark liver-green or black. In consistence they may be as hard as mahogany, or as soft as firm cheese so that often they can be broken up by the fingers in the ducts. Those which are softest consist entirely of bile-pigment, and may be confined to the smaller ducts in the liver. There may be but one large ovoid, barrel-, or pear-shaped stone occupying the gall-bladder, or many hundreds of wedge-shaped or rounded forms may be found in it. In the unusual cases where the calculi are found in the liver they are always small; the calculi which are found blocking the common duct are certainly of cystic origin. Though most calculi float in water after they have been dried by removal from the body, some are found to be heavier than water when passed from the bowel. Those which consist mainly of cholesterin always float.

Etiology.—The disease is rare before the twentieth year of life, the majority of cases occurring between forty and sixty.

Women are affected between three and four times more frequently than men, and a very large percentage of the cases occur in women who have borne children. Sedentary habits and occupations which interfere with the full play of the abdominal muscles, tight-lacing, and ptosis of the liver, or Glénard's disease, and all causes which tend to favour stagnation of the bile, are important etiological factors.

Diet is operative in many cases, the condition being common in persons who indulge heavily in farinaceous foods and fats, and less likely to happen in meat-eaters, because in these there is abundant formation of bile-salts, which Robson believes tend to keep the cholesterin in solution. Though cholelithiasis is often stated by writers to be the result of over-indulgence in food generally, the writer has often witnessed the most inveterate form of the affection in spare eaters who also partook of a minimum of fluids and who seldom ate butter or fats. This type of feeder is very prone to suffer from gall-stones, and of his trinity of dietetic errors the deficiency in liquids is probably the chief factor.

The condition has also been supposed to be a legacy left by various infective diseases, especially typhoid fever; and of late years the close connection between cholelithiasis and this disease has been frequently demonstrated.

Symptoms.—It is a well-recognised clinical fact that the gall-bladder is often found full of calculi after death in persons who have never exhibited any symptom of gall-stone during life, and it is calculated that they are to be found in about one body of every twelve examined if all ages be included; recent German statistics prove that they are found in about one in every four cases after the age of sixty. This is but another way of stating that it is only in a small majority of cases of cholelithiasis that the calculi betray their presence by any symptoms.

Hepatic or Biliary Colic.—Though jaundice may be the only symptom produced by the impaction of a large calculus in the common duct, or distension of the gall-bladder when the cystic duct is blocked, there is usually more or less pain, and the entrance of a stone into any of the larger ducts from the gall-bladder usually causes intense suffering of a characteristic type.

This comes on suddenly, usually without any apparent cause or after some slight muscular movement, or after a heavy meal. It is of such excruciating severity that the patient often feels compelled to cry out during the violence of the paroxysm, and he wriggles about and tries every possible posture or attitude which he hopes may mitigate the agony of his suffering. The pain starts in the right hypochondrium under the costal arch and radiates to the epigastrium, round the side, and upwards towards the right scapular region,

though occasionally the pain may be greatest in the left shoulder.

Vomiting is an almost constant symptom; it usually sets in after the pain has lasted for some time, and it may be severe and continuous, not unlike sea-sickness. Where the stone is small and lodged in the intestinal end of the common duct, the advent of emesis, by producing a general muscular relaxation, may terminate the attack by permitting the obstruction to enter the duodenum. This event will be heralded in by the abrupt cessation of all pain. More or less collapse usually attends the vomiting, and the pulse is weak and quick. Fever is usually absent at first, but some cases are ushered in with a smart rigor, followed by profuse sweating; at a later stage the fever may rise to 104° or more, probably owing to the absorption of some retained products.

During the seizure tenderness is always present over the liver and about the region of the gall-bladder, and it usually remains for a considerable time after the attack has passed away. During the height of the paroxysm, though the pain is continuous, it is liable to slight brief remissions, and some intelligent patients, without any knowledge of the cause of the pain, will liken it to what they think might result from some hard body being pushed through a narrow channel or aperture.

The duration of the paroxysm is most variable: it may last for a few hours or it may be prolonged into several days, when often it might be described as a series of rapidly recurring paroxysms with practically only slight moments of partial relief.

The continuous pain is probably caused by the stretching of the walls of the duct, and the acute exacerbations or colic to the peristaltic exaltation. As already stated, the *abrupt* or sudden termination of the pain is indicative of the passage of the stone, but more frequently the pain *gradually* and slowly subsides, leaving considerable tenderness and a sense of soreness behind, as it becomes for the time wedged in the duct. Jaundice supervenes after some hours, or it may be after one or two days, according to the completeness of the plugging; but it does not appear when the obstruction is in the cystic duct or when the stone slips back into the gall-bladder or is passed speedily into the duodenum. The advent of marked yellowness of the skin and the appearance of bile-pigment in the urine may be taken as conclusive evidence that the common duct is plugged, and the motions will be found to be white, though the first evacuation passed after the seizure is usually normal in colour.

The gall-bladder is seldom markedly distended in acute attacks; and though its muscular contractions may produce a hard tumour, this is not easily demonstrated owing to the

great tenderness. In blocking of the common duct, it must always be remembered, the flow of bile into the gall-bladder soon ceases; and though there may be a very slight temporary distension from retained bile, this is gradually replaced by pure mucus, and the receptacle usually contracts or shrinks in volume, so that it may be accepted as a safe clinical rule that in obstruction of the common duct there is no enlargement of the gall-bladder present.

As long as the blocking continues complete in the common duct, the jaundice deepens and remains persistent; the liver enlarges and becomes tender, and the spleen swells. This condition is likely to remain till one or more fresh paroxysms occur, during which the stone is driven into the intestine. The point at which blocking most frequently occurs is at the intestinal orifice of the duct.

Permanent blocking of the common duct, though rare, may, however, occur and be *complete*, in which case there will be great dilatation of all the ducts throughout the liver, which finally become filled with clear mucus. The jaundice is lasting and intense, and fever is absent; and though in rare cases there may be some distension of the gall-bladder, in the great majority of the cases it is contracted.

Permanent blocking is more frequently *incomplete*, in which case bile is usually present in the motions, the jaundice is intermittent or variable in intensity. When the skin-coloration is deep the motions become white. Fever of the malarial type is always present at intervals, owing to the introduction of bacteria from the bowel; there is great distension of the ducts; the liver and gall-bladder are generally normal in size, though there is splenic enlargement. This condition may last for many months, or for some years, but at any time there may supervene a suppurative cholangitis or cholecystitis (see page 431), which may end in perforation or hepatic abscesses and death.

Calculi confined in the cystic duct may produce colicky pain identical with that resulting from blocking of the common duct, but *jaundice does not occur*. The gall-bladder is at first more or less distended with bile; this soon is replaced by a clear thin mucous liquid, and the distension gradually increases till a tumour is formed, which may grow to huge dimensions in rare cases, when it is known as dropsy of the gall-bladder, and has even been mistaken for an ovarian cyst.

The following are the chief characteristics of the tumour caused by enlargement of the gall-bladder. The swelling has a smooth surface and is usually pear-shaped, but often the blunted lower extremity cannot be definitely palpated. It conveys the idea of the form of the old-fashioned egg-shaped lemonade-bottle depending from the liver under the costal

arch with its neck behind the ninth costal cartilage when the liver is not enlarged or depressed. Upon deep inspiration the tumour will be found to descend with the liver. Fluctuation can sometimes be made out when the parietes are thin and the organ not too tightly distended, but percussion gives little aid in the examination. Occasionally, upon bimanual exploration with the tips of the left fingers in the loin and the right hand grasping the tumour, the gall-stones, may be made to convey a dull crepitus to the examiner's touch, and a similar phenomenon has been sometimes detected upon auscultation.

When the lower edge of the liver has descended owing to its enlargement the tumour may be depressed below the level of the navel, and it is usually easy to make out a marked gap or sulcus between its border and the rotundity of the tumour, which may be visible upon watching the action of the diaphragm. Usually the distended organ can be freely moved from right to left by palpation of its base, though the neck is fixed; yet when inflammatory adhesions have formed, the tumour becomes very tender, hard, and firmly fixed, and may not move on deep inspiration.

The physical signs will vary considerably upon the supervention of acute cholecystitis; and when empyema of the organ occurs there may be great enlargement from the formation of pus in its cavity. Sometimes the gall-bladder becomes calcified owing to the deposition of lime-salts, in which case it may shrivel into a small mass of stony hardness. In a large number of cases the organ shrinks and becomes impalpable after the absorption of the retained bile and mucus which had caused its previous distension. So common is this latter result that in the majority of gall-stone cases the gall-bladder will be found contracted, whilst its enlargement is often strongly suggestive of malignant disease of the head of the pancreas.

It is very rare for an attack of biliary colic to cause death, but this result has happened either from the collapse caused by the incessant vomiting or from rupture or perforation of the ducts or gall-bladder. These latter complications are more likely to occur where the blocking has been of long standing, when a host of other sequelæ or serious results may supervene. The pressure of the calculus may cause local ulceration of the ducts, which will end in perforation and the escape of the stone into the peritoneal cavity, causing perforative peritonitis; or adhesions having been previously formed, the stone may enter an abscess-cavity or discharge directly into the stomach, small intestine, or colon, portal vein, pleura, or kidney, leaving a permanent fistulous communication between these parts and the biliary passages or gall-bladder.

Where adhesions and ulceration occur, the most common result is a localised abscess in the abdominal walls and the discharge of the stone through the skin-wound externally, often about the umbilical region. From the fistulous opening bile and calculi may continue to discharge for indefinite periods.

Obstruction of the bowel, which may prove fatal, occurs in a considerable number of cases where the stone ulcerates into any portion of the intestinal tube; and it is a remarkable fact, of which no obvious explanation seems forthcoming, that this may result from the entrance of even small calculi, whilst sometimes very large ones are passed without much difficulty.

Description need not be given of other complications, already incidentally referred to in the previous pages, as hepatic abscess, suppurative cholangitis and cholecystitis, pyæmia, obstruction of the bowel from adhesive bands produced in the region of the gall-bladder, or of general or localised peritonitis caused by extension of the inflammatory action from the bile-passages. Dilatation of the stomach from old adhesions is not very uncommon, and cancer of the gall-bladder, biliary ducts, or liver frequently supervenes in cases of long standing. Permanent stricture of the ducts may follow in some cases after the expulsion of calculi.

Death sometimes follows from hæmorrhages, which may occur in any region or from the mucous surfaces, owing to the alteration produced in the blood by the persistent jaundice; and the tendency to hæmorrhagic oozing after operations is a source of serious danger. The itching, which is often most persistent and intolerable all over the cutaneous surface, has a like origin in the blood-condition.

Diagnosis.—*Renal colic* is only likely to be mistaken for an attack of gall-stone colic when the calculus has entered the right ureter, and then the following points will serve to distinguish it from an hepatic paroxysm: the seat of the pain is in the flank, it radiates downwards in the direction of the ureter and into the right testicle, which becomes tender and retracted; it may be complained of also along the inner side of the thigh; blood often appears in the urine, and micturition is frequent. In all doubtful cases the fæces should be carefully examined for calculi, the patient being directed to pass his motions into a nightstool half filled with any warm disinfecting liquid; upon passing the liquefied motion through a moderately fine-meshed sieve the calculi will be easily detected. They usually sink in water, but after drying they readily float. By a careful examination of the surface of the stone for facets a diagnosis of the probable presence or absence of others may be made. The supervention of even a trace of jaundice is of value in the diagnosis of hepatic colic.

Hysteria may sometimes simulate the paroxysm, but the other manifestations of hysteria will be found if looked for, and there will be no rise of temperature, collapse, nor jaundice, nor palpable tender tumour.

Movable or *Floating kidney* on the right side can usually be excluded by carefully palpating the abdomen, when the tumour can generally be demonstrated by bimanual examination ; the pain in some cases instantly disappears by inverting the patient.

Acute dyspepsia is speedily relieved by emesis ; but it may be present and may be the determining or exciting cause of the attack of gall-stone colic, in which case the diagnosis is obvious after its removal by vomiting.

Gastralgia occurs only in neurotics. The seat of the pain is over the stomach, and to the left or in the middle line ; fever, jaundice, and chills are absent, and marked tenderness and enlargement of the gall-bladder do not exist.

Appendicitis, or *Appendicular colic*, is distinguished by the swelling and circumscribed tenderness over M'Burney's point ; but if acute cholecystitis has supervened on gall-stone colic the diagnosis may be difficult, and can only be settled by a critical examination of the history of the case, or by an exploratory incision, especially as in both conditions jaundice may be absent.

Malignant disease of the liver, pancreas, ducts, or gall-bladder can often be only excluded in chronic cases after watching the progress of the affection for some time. The steadily progressive emaciation and the slow deepening of the jaundice, with the markedly continuous type of both the pain and fever, when these are present, are almost pathognomonic of cancer. The presence of obvious gall-bladder tumour is strongly in favour of the more serious disease, as insisted upon by Mayo Robson, especially when the head of the pancreas is affected. The appearance of hard nodules in the liver and of ascites renders the diagnosis certain. Mistakes are often made by grounding the diagnosis upon the duration of the condition : though cancer of the liver rarely lasts more than a year, it can never be safely excluded by a time-limit in the most chronic cases, since it is often a sequela of old gall-stones.

A history of previous attacks is too often regarded as of vital importance in diagnosis ; all the affections in the above list are liable to recur repeatedly.

Prognosis. — The different points have been indirectly referred to in the description of the complications ; the prognosis, though good in simple uncomplicated cholelithiasis, may in any case become grave upon the supervention of cholangitis, cholecystitis, or other serious complication. The introduction of the brilliant surgical methods of treatment

during late years has enormously brightened the prognosis of the disease, and prevented recurrences.

Treatment.—The details of the management of cholelithiasis have been described in the author's 'Dictionary of Treatment' (4th ed.), pp. 303–308. Only the principles of treatment need be here referred to. Pain during the paroxysm calls for morphia hypodermically, or for chloroform in very severe cases. Poultices, fomentations, and hot baths are advisable as adjuvants.

Starches and fats should be used sparingly, alkaline diluent drinks freely partaken of, and active exercises indulged in daily after the attack, with the view of prevention of recurrences.

Agents administered with the view of dissolving the stones are all of doubtful value, but many firmly insist upon the value of large doses of olive-oil. Unquestionably, calculi are often expelled after large doses of calomel followed by castor-oil.

Surgical measures are resorted to with increasing frequency and success every year. The following are the conditions most commonly met with in which operation is considered justifiable:

(1) In gall-stones impacted in the common duct associated with persistent jaundice and repeated attacks of pain; the period during which medical remedies are to be persevered with must be decided upon by the severity of the general symptoms and condition of the patient, but it is safe to say that the mistake generally made is in delaying too long; (2) when attacks of hepatic colic, with or without jaundice, occur repeatedly; (3) in enlargement of the gall-bladder without jaundice; (4) in enlargement of the gall-bladder with jaundice, where malignant disease is not probably present; (5) in all suppurative conditions of the gall-bladder; (6) where infective cholangitis has occurred; and (7) in cases where biliary fistulæ have formed.

The usual operation in ordinary cases is *cholecystotomy*, or opening and draining of the gall-bladder. *Cholecystectomy*, or removal of the organ, is only justifiable when the ducts are free from all calculi, in malignant disease, and in cases where, from phlegmonous inflammation, empyema, or calcareous degeneration, the gall-bladder has become useless or the duct strictured. *Choledochotomy*, or incision of the cystic or common duct, is undertaken when *cholelithotrity*, or crushing of the stone in the unopened duct, is impossible. In cases where clearing of the ducts is impossible, or where the common or cystic ducts are strictured, the gall-bladder must be short-circuited—*i.e.* an anastomosis much be established between it and the intestine; this operation is known as *cholecystenterostomy*

The mortality of gall-bladder operations, taken all round, in gall-stone cases, probably is considerably under 5 per cent. Lilienthal's recent list of forty-two primary cholecystectomies had only a single death; this was the result of a streptococcus infection, which was present before the operation. The statistics of the last 500 cases of gall-bladder operations by W. J. and C. H. Mayo include 272 cholecystotomies, with a mortality of 1·47 per cent. Mayo Robson states that the mortality of cholecystotomy in the early stages of cholelithiasis is now less than 1 per cent.

Massage of the abdominal wall as a means of forcibly pressing the stone onwards into the duodenum, should never be resorted to, owing to the danger of injury or rupture of the gall-bladder or ducts.

GANGRENE

Also popularly known as Mortification, is the term used to signify the condition of an organ, tissue, or limb in which there is complete cessation of the functions of organic life. It is synonymous with the *death* of the portion of the body involved in the gangrenous process.

The subject is chiefly of surgical interest, but the causes which tend to produce the death of circumscribed portions of the body often belong to the province of the physician. Intense inflammatory action and the complete cutting off of the blood-supply of the part may eventuate in the death of the injured or starved tissue. Under the headings of 'Arteritis,' 'Embolism,' and 'Thrombosis,' the latter cause will be dealt with. The local asphyxia which constitutes the most obvious feature of the symmetrical gangrene met with in cases of Raynaud's disease belongs to the same category.

The condition of the blood and tissues in diabetes, pneumonia, typhoid and typhus fevers, measles, and scarlatina sometimes favours the advent of gangrene. Ulcerative or malignant endocarditis is a not uncommon cause of the death of a limb by means of the infective vegetations detached from the cardiac valves which cause plugging of the chief arterial supply.

Gangrene of the lung and stomatitis of the gangrenous type will be described under their own headings.

GASTRALGIA, or GASTRODYNIA

This is the name given to a symptom met with in several diseased conditions of the stomach or of the gastric nerves. The characteristic features of this variety of severe paroxysmal pain have been described upon page 335. When occurring

without obvious organic disease, it is to be regarded as a true gastric neurosis. But it may be the predominant symptom of a serious organic lesion of the stomach, as ulcer, cancer, or gastritis, whilst it is often met with in its typical form in the gastric crises of locomotor ataxia.

The chief differential points have been already enumerated under 'Dyspepsia.'

GASTRECTASIS

This is the name given to dilatation of the stomach, which presents itself in several well-marked forms. The common condition of enlargement of the stomach, which is found in gross feeders and in those who live upon vegetable diet and drink large amounts of fluid, should not be included under the above term. Such cases of so-called '*megalogastria*' exhibit no abnormal digestive symptoms, as the motor power of the stomach is intact and there is no obstruction.

Etiology and Pathology.—Gastrectasis or Ectasy implies always dilatation of the organ of a more or less permanent character, associated with insufficiency of motor power. Acute dilatation is a rare disease which will be referred to afterwards, as the present article deals chiefly with the chronic condition. The essential feature in all typical cases is *the constantly over-filled condition of the dilated organ, with the retention of the ingested food for an abnormally long period.*

The cases naturally divide themselves into two well-marked types, corresponding to their origin: (1) The *Atonic* form, in which the primary mischief is loss of the muscular power (atony) of the gastric coats to drive the food into the intestine within the normal period after ingestion. This may be the direct result of continuous gluttony, or the ingestion of large amounts of very diluted liquid foods at such short intervals that the over-worked stomach-muscle fails; or it may be caused by a primary atony of congenital origin, or a degeneration of the muscle following such conditions as chronic gastritis, cancer, dyspepsia, anæmia, tuberculosis, and the infectious fevers.

(2) The *Obstructive* form. This type of ectasy is produced by narrowing of the pylorus, or more rarely by obstruction in the duodenum. The pyloric stenosis may be the result of the cicatrisation of an old ulcer in the duodenal end of the stomach, or it may be caused by a cancerous growth at the sphincter, by pyloric hypertrophy, fibroid thickening or other innocent growth, or by adhesions between the pylorus and neighbouring organs, as the gall-bladder, &c. The obstruction may be caused by a gastroptosis which prevents the emptying of the stomach through kinking of the duodenum near the pylorus.

Congenital hypertrophy, leading to narrowing or stenosis of the pylorus, may produce ectasy, and it is more frequently the cause of death in young infants than is usually recognised. The writer believes that the cases of congenital stenosis, without hypertrophy, are akin to those which he has witnessed of a narrow or impervious state of the bile-ducts causing death in several infants of the same parents.

In all these cases of ectasy depending upon pyloric obstruction the muscular power of the stomach-walls may be greatly increased, as hypertrophy is common ; but motor insufficiency must be recognised as always present, since the increased motor power is unable to overcome the mechanical impediment. At a later stage atony supervenes from degeneration of the muscular fibres, as the stomach becomes distended into a flaccid bag.

Symptoms.—Of the gross or obvious diseased conditions of the abdominal organs, few are so easily recognisable with certainty as a case of well-marked dilatation of the stomach, and few are so frequently entirely overlooked. The following description of symptoms and signs may be taken as applicable to a typical case of dilated stomach following the cicatrisation of an old pyloric ulcer.

In the first place there is a difficulty in defining the limits of normal variation in the dimensions of an organ so subject to changes in volume as is the healthy stomach. Though the lower margin of the healthy stomach is, according to Meltzing, much below the level of the navel when distended with food, this is quite impossible of demonstration by the ordinary methods. It may be accepted that when the lower limit is obviously below this level more or less pathological dilatation is present, if the upper part of the stomach be in its normal position.

Of far more importance, however, than the mere bulk of the stomach is the significance of even a moderate distension at a period after eating, when the organ should be quite empty.

The history of the case will usually show a gradually increasing difficulty in digestion, with considerable discomfort and fulness after food, which ultimately tends to become continuous ; eructations, often of an offensive kind, with pyrosis, have been experienced for a considerable period before advice is sought. In typical cases there may be a clear history of a gastric ulcer long before, which had evidently healed, leaving the patient quite well for many months or several years.

There is one point in the history of these cases which is occasionally mentioned by the patient, and which should be always sought for in eliciting the details of his symptoms, and if present it almost settles the diagnosis at once. This is

the fact that he has at some time or other recognised in his vomit some particles of food swallowed several days before. If, with this discovery, there be a clear statement that he has often vomited, upon a single occasion, four or more pints of gastric contents, the condition is evidently one of dilatation. These points will be again referred to when considering the progress and symptoms of the disease; in the meantime the steps in the physical examination may be detailed.

Inspection.—In thin subjects this may be conclusive: as the patient lies upon his back the outline of the stomach may be visible through the relaxed abdominal walls. If the organ be much dilated it descends somewhat, and its smaller curvature may be visible in the transverse depression at the upper part of the epigastrium, whilst its convex lower margin may extend to near the pubis. But it is necessary to remember that a simple *gastroptosis* may simulate this condition; but in ptosis the position of the displaced organ is more vertical, the depression above is better marked, and careful inspection will show that both curvatures have correspondingly fallen, and the entire bulk of the organ is not materially enlarged. These points become more evident in the standing position, and after inflation of the stomach by air or gas.

Inspection will often show the presence of waves of peristaltic movement in the protrusion corresponding to the stomach—the so-called peristaltic unrest of Kussmaul. These are usually most obvious along the greater curvature, passing from left to right, and are best examined by permitting the light to fall on a level with the patient's trunk, so that the shadows greatly intensify the surface-irregularities produced by the wave-like movements; they can always be increased by kneading or flipping the abdominal wall briskly. The administration of an effervescing draught will also make the waves more obvious.

Palpation.—This is a valuable method, but usually much less so than inspection. It should be gentle and deliberate. The ulnar portion of the hand placed below the xiphoid may sometimes mark out the upper curvature when other methods fail. The bimanual plan (clapotage), with a hand on each side of the abdomen, will elicit splashing or succussion sounds over a large area, when sudden pressure is made in the direction of the middle line. Riegel rightly insists upon the worthlessness of this method, unless the importance of the time at which it is elicited in reference to the ingestion of food or liquids be carefully borne in mind. In dilatation of the stomach it can be produced seven hours after a full meal and two hours after a test breakfast, and the splashing can be heard and felt over a large area, extending below the umbilicus. It can always be elicited in healthy subjects soon after a meal, but

over a comparatively limited area. Too much importance is attached to the mere acoustic portion of this test by Riegel and other authorities: the sensation of fluctuation, in the writer's opinion, is of much more importance than the sound which is produced ; by the sensation of fluctuation, a correct idea may be formed of the bulk of the distended stomach, even when little or no sound is produced.

It fails to differentiate between ptosis and dilatation, unless the latter is very extensive, and it must always be remembered that a dilated colon may mislead.

Percussion.—This method of examination by itself is often of little value, but when combined with others it is most useful in the recumbent posture. The lower and upper borders of the stomach are often mapped out with difficulty and uncertainty, owing to the resonance of the colon ; but in dilatation, when the patient is in the erect posture, the lower margin of dulness can usually be located below the level of the umbilicus, and the dulness disappears upon resumption of the horizontal position. By mapping out the level of dulness in the erect posture and removing the contents of the stomach the dull area becomes clear ; upon pouring in fluid through the syphon the dulness reappears and gradually extends *upwards* and slightly *downwards*, as the dilated organ is filled. Little aid is obtainable by the method of auscultatory percussion, and less by simple auscultation, though sometimes the fizzling sounds produced after the administration of an acid followed by an alkali may be heard over an area so extensive as to suggest dilatation when the stethoscope is applied.

Syphonage.—Often by passing a soft rubber tube and drawing off the gastric contents the nature of the case is at once obvious from the mere bulk of the fluid evacuated. The nature of the gastric contents will be referred to later on.

Inflation of the stomach by air can be easily accomplished by attaching the nozzle of an ordinary enema-syringe to the extremity of the syphon and pumping in briskly as much air as can be borne without distress. In this way the dilated organ becomes ballooned, so that its dimension, borders, and position in the abdominal cavity are demonstrated. By this method the outline of the *upper* curvature is rendered evident, so that dilatation is distinguished from ptosis or other form of dislocation ; hence its great value.

Where from any cause the tube is contra-indicated, the distended organ can be inflated by the administration of a teaspoonful of sodium bicarbonate dissolved in half a tumblerful of water, followed by a similar dose of tartaric acid. This method is open to one serious objection rarely mentioned: in severe pyloric obstruction the stomach is usually distended by fluid, except after vomiting, and the writer has seen the carbonic-acid test thoughtlessly applied under these circum-

stances, and entirely wrong conclusions arrived at from the result. The stomach should be always emptied first.

The writer has determined the lower border of the stomach by causing the patient to swallow a small polished steel ball with a silk cord attached, and then located its position by the Röntgen rays with the patient in the erect posture.

Recently a decided advance has been made in the physical diagnosis of the size and state of the stomach by the use of the X-rays, after the administration of an insoluble bismuth-salt. The subnitrate throws a deep shadow under the X-rays, and the drug may be mixed with food or given in capsular form. In this way, upon the fluorescent screen the boundary-lines of the stomach may be demonstrated in a way hitherto impossible, and already the plan has been made available for testing the rapidity of the digestive process by enclosing the bismuth-salt in envelopes of goldbeater's skin or thin animal membrane, and watching the time required for the solution of the envelope. The process has also been employed for the detection of impalpable malignant growths, since these have been found to cause obvious changes in the picture upon the screen, when bismuth-food is being digested or churned about through a stomach whose walls are infiltrated with new-growth at certain parts.

The symptoms will vary with the degree of completeness with which the pylorus is narrowed. In all cases there will be more or less dyspepsia, gastric uneasiness, and belching of flatus, with acidity. Appetite is often unimpaired and may be excessive, but thirst is always a marked symptom.

Constipation is almost invariably present in an obstinate form in severe cases, and the amount of urine is materially diminished, and it is usually alkaline ; the skin is dry and harsh ; the nutrition of the body becomes seriously affected, and emaciation sets in and may be very marked. In cancer cases, which are not now under consideration, the wasting becomes extreme.

Vomiting sooner or later sets in, and becomes the dominating feature in the clinical picture ; its characters are of great importance. At first it occurs at irregular intervals after some exacerbations of the dyspeptic symptoms, but even in early stages of the affection it comes on late after eating, and it always affords considerable relief. As the dilatation becomes established, vomiting occurs at fairly regular intervals ; it may be absent for days, however, when several pints of sour, bitter, and offensive liquid are ejected after much pain and discomfort. In the vomitus, remnants of the food are sometimes recognised which had been taken several days before.

Occasionally one sees a totally different type of vomiting in advanced cases where the stomach has become a mere fibrous bag unable to empty itself. Here there may be the

frequent vomiting of small quantities of yeasty liquid which come up without effort—a phenomenon somewhat akin to the overflow of a distended bladder. In these cases the tube may syphon off enormous quantities of liquid and some food swallowed weeks before.

The gastric contents, when allowed to settle, separate into three layers—the lowest containing imperfectly digested fragments of solid food, the middle of a dirty grey colour, on the surface of which floats a yeasty, frothy, brown layer. By watching the glass vessel in which the offensive liquid is placed, bubbles caused by active fermentation are usually visible.

Butyric and other organic acids are present and free hydrochloric is usually in full amount. Various bacteria, yeast-cells, and sarcinæ are found, with undigested muscular fibres and other food-elements.

Bile will exist in large amount in those rare cases where the obstruction is beyond the entrance of the bile-duct in the duodenum.

Nervous symptoms are common, as headache, vertigo, insomnia, &c. A group of symptoms resembling tetany is sometimes observed, and in rare cases this *gastric tetany* will present all the features of the typical disease, with, however, this important and serious difference, that it is nearly always fatal.

There is a sudden contraction of the muscles of the upper extremity, in which the fingers are flexed at the knuckles whilst extended at the phalangeal joints; the tips are approximated, forming the so-called accoucheur's hand; the feet and toes are also affected. Trousseau's sign can be elicited—*i.e.* the attack can be repeated again before it finally passes off, by pressing upon the nerves or blood-vessels of the forearm. Chvostek's sign is present—a tap over the facial nerve will cause active contraction of the facial muscles; the electrical irritability of the motor nerves is increased—Erb's sign; and the mechanical and electrical excitability of the sensory nerves are increased—Hoffmann's sign. (See also under 'Tetany.')

Attacks of tetany have been reported in several cases after lavage. The writer has witnessed a first attack which proved fatal where the stomach-tube was not employed.

There is much speculation about the pathology of gastric tetany. Kussmaul, who first described it, attributed it to dehydration of the tissues; others regard it as a reflex phenomenon. The recent view is that it is of toxic origin, and certain changes found in the brain-cells observed by Ferranini support this theory. That auto-intoxication from the absorption of products formed in the dilated stomach is the probable cause receives confirmation from a case which the writer saw recently with Dr. Trimble. The tetany, which was

well marked, was associated with active delirium, and the patient died comatose.

When the gastric dilatation is the result of a *carcinoma* of the pylorus which causes obstruction, the symptoms are the same as those mentioned; but the size of the distended organ is rarely so great as in benign pyloric stenosis, as death will occur at a comparatively early stage. There is great cachexia and more profound wasting; a tumour can usually be felt, near to the umbilicus; the appetite is much impaired, and the gastric contents will usually show absence of free hydrochloric, with much lactic acid.

In those cases of ectasy originating in atony, and characterised by progressive muscular insufficiency, the symptoms are much milder than those just described as belonging to an advanced case of pyloric stenosis; but they are the same in kind, though not continuously progressive in most cases. The dilatation increases with indiscretions in diet.

The physical signs are usually less marked, but food will be always found in the stomach long after the organ should have emptied itself into the duodenum.

Where motor insufficiency exists alone, without dilatation, the case does not fall under the category of gastrectasis, but rather belongs to the gastric neuroses.

The progress of gastric dilatation depends upon the primary cause; in cancer, of course, it is limited by the progressive nature of the fatal disease. In those cases arising from atony the condition may last a long lifetime; but by judicious dieting the malady may be entirely removed before permanent dilatation has become established. Even when obstruction is almost complete at the pyloric ring and the stomach enormously dilated, the patient, by daily lavage, may enjoy a fair degree of health for years. The writer saw a case fifteen years ago in which he introduced sixteen pints of water into the dilated stomach, and syphoned it off. The patient lived in comparative comfort by daily washing out the organ himself; he has recently died from a sudden attack of tetany, accompanied by delirium and coma. Such a distension, with the view of measuring the capacity of the dilated organ, he now thinks is unjustifiable, as it might be followed by rupture.

Acute dilatation of the stomach is a very rare condition first described by Fagge. It is liable to end in a fatal issue. The writer was fortunate in seeing one case, and it ended in recovery. Vomiting suddenly sets in after some acute dyspeptic symptoms, and enormous quantities of fluid are ejected; but the stomach rapidly fills up again and becomes paralysed by the distension. There are various theories invented to explain the condition, but its pathology is unknown; the source of the vomited liquid must, however, be a secretion or exudation from the gastric mucosa. The condition has been noticed

as a complication or sequela after surgical operations. More numerous are the cases of acute dilatation which have followed an act of gross over-feeding or gluttony in children.

Pathological Anatomy. — The anatomical changes will vary with the primary disease causing the dilatation or ectasy. In cases of pyloric stenosis there is usually much hypertrophy of the musculature in the distal end of the stomach, whilst atrophic changes are found along the great curvature; but in long-standing cases, and in atonic cases, the entire muscular tissue may be thinned out so as to be recognisable with difficulty. In other cases hypertrophied bundles of muscular tissue may be visible, alternating with thick bands of fibrous tissue; and occasionally, where the organ is not much increased in capacity, a sclerotic condition of all the coats may be met with. The mucous coat is much atrophied when cancer is the primary disease, and evidence of gastritis is common in all cases from the irritation produced by the retained fermenting contents. Occasionally, as in one case recently seen by the writer and diagnosed before operation, an hour-glass contraction of the stomach is found in association with the dilatation.

Often the pylorus is found dragged downwards by the weight of the distended organ, so that the kinking of the duodenum may cause in atonic cases as complete obstruction as is to be seen in fibroid stenosis of the pylorus itself. This is the explanation of those cases of ectasy which supervene upon ptosis of the stomach in Glénard's disease.

Diagnosis.—The various points in the recognition of the dilated stomach have been already detailed under the physical signs and symptoms. The greatest difficulty occurs in the differentiation between dilatation and simple gastroptosis. If the smaller curvature be in its normal position and the lower border of the stomach be obviously below the umbilicus, and food be found in it seven hours after a full meal, dilatation is certain. In doubtful cases the organ should be inflated, when both curvatures may be demonstrated. There is practically no difficulty in the diagnosis when both conditions occur together; the presence of food long after a meal, even with great descent of the small curvature, proves the case to be one of ectasy. Whether the ptosis is the primary lesion can usually be determined by the history of the case, as the liver and kidney are usually displaced also in ptosis.

The discrimination of the class to which the dilatation belongs—*i.e.* whether it is *atonic* or *stenotic*—is often difficult. As a rule, the larger stomachs are caused by stenosis and the visible strong peristaltic movements only occur in them, and there is often a history of old ulceration. In the use of the tube during lavage the experienced physician can easily judge of the propulsive power of the stomach, and Riegel

points out that the water flows into a flaccid atonic stomach with greater ease than into one whose muscular walls are hypertrophied through pyloric obstruction.

The diagnosis between *benign* and *malignant* stricture of the pylorus is not usually difficult. It has been already incidentally referred to. In practice the duration of the ailment settles the question easily in many cases. Absence of free hydrochloric acid, the presence of a tumour, marked emaciation, and cachexia are characteristic of cancer.

The method of illuminating the stomach—gastrodiaphany or electric transillumination—with the view of demonstrating its boundaries is too complicated for general use, and moreover the results appear to be open to misinterpretation; hence it has not been referred to as a part of the physical examination.

The usual tests for the determination of the rate of absorption from the walls of a dilated stomach have not been mentioned; they seldom prove useful. Iodide of potassium, given in a gelatin-capsule on an empty stomach, should appear in the urine in seven to fifteen minutes; in dilatation it may not appear sooner than an hour or more.

Salol, and more recently iodipin, are used to test the motor power of the stomach; these drugs, after being swallowed, remain unchanged in the stomach, but on entering the duodenum they are split up by the alkaline secretions of the liver and pancreas. Any marked delay in their excretion indicates retarded passage of the food out of the stomach. Salol is unreliable, unless used by Huber's plan—*i.e.* 16 grs. are given with the midday meal, and the bladder is emptied twenty-seven hours afterwards. If salicyluric acid be found in the urine three or four hours later, there is motor insufficiency. Iodine can be detected in the saliva in a quarter of an hour after iodipin has been swallowed in a capsule under normal conditions; but if motor insufficiency exist, its appearance may be delayed for three-quarters of an hour or more.

Both these tests are inferior to Leube's—*i.e.* the stomach-tube is used seven hours after a meal of a plate of soup, beef-steak, and bread. Under normal conditions of motor power the stomach should be found empty.

Hour-glass contraction of the stomach may sometimes simulate dilatation; it will be referred to under 'Gastric Ulcer,' and, as just mentioned, both conditions may exist together.

Prognosis.—In detailing the course and progress of dilatation of the stomach the prognosis has been incidentally dealt with. The chief point in forecasting the outcome is the nature of the primary disease. A fatal result will always follow cancer within a year or two, though generally the issue can be retarded by operative procedures. Atonic cases will usually yield to proper dietetic treatment if instituted early. Benign stenosis is incurable save by surgical measures,

which usually give brilliant results; but enough has been said to show that dietetic treatment and lavage may keep the patient alive in a fair degree of comfort for a very long period.

Treatment.—In atonic cases, in their early stage, a carefully regulated diet of semi-solid food, given in small quantities very frequently repeated, is all that is necessary to effect a cure. It is in most cases undesirable to place the patient upon a so-called strictly dry diet. Solid food increases the demands made upon the enfeebled musculature, but finely minced meat and chicken may be permitted in small amounts. Milk can be given in fair quantity daily, if the dose be limited to a large spoonful, or small wineglassful, at a time; strong jellies and soups may be administered upon the same plan. Vegetables, alcohol, tea, and coffee are usually harmful.

Often very striking results may be observed as soon as all gross feeding is prohibited. In severer cases the bulk of liquids given by the stomach must be considerably diminished, and this can be compensated for by large rectal injections of weak saline solution or nutrient enemata.

Where there is already evidence of great dilatation, with yeasty vomiting and gastric distress from long retention of the stomach-contents, it is useless to hope for any marked reduction in the dimensions of the dilated organ by diet alone, whether the cause be a neglected atony or pyloric stenosis.

In these cases lavage must be resorted to without delay; and it is a good rule to insist upon a daily washing-out of the stomach in every case where food is found in the organ seven or eight hours after a full meal. The patient, seated in a chair, has the soft rubber stomach-tube passed through the gullet into the gastric cavity, and the contents syphoned off, after which tepid water is poured into the funnel, and by elevation of the latter above the level of his head the fluid is permitted to flow in to the extent of about a pint at a time. Upon depressing the funnel this is again drained off, and the operation repeated till the washings become clear. Where there is much stagnation and fermentation various antiseptics have been recommended. The best of these (and the least liable to do harm) is a weak permanganate-of-potassium solution, enough to stain the water a decided pink colour; creosote is also very valuable if carefully used, but it should not be left in the hands of the patient without strict supervision.

After a few sittings the patient can usually perform the operation upon himself with the assistance of a nurse. Food may be administered almost immediately after the washing process has been completed, and where the capacity of the dilated organ is very great it is often advisable to pour in a

pint of warm soup before withdrawing the tube. Though it may be necessary to use a gallon or more fluid during the operation, the patient and nurse should be instructed not to permit more than a pint to be present at any moment in the stomach. The tube may be pushed home or slightly withdrawn occasionally, or the patient may shake his body from time to time, or a little more fluid may be poured in, should the outward stream cease to flow.

Riegel advises that lavage should be performed in the evening before supper, so that the stomach is secured rest during sleep ; and if much food be found in it in the morning he advises an additional morning wash-out. Few patients will submit to this, and most victims of dilatation find that they can get more comfortably through their daily work after the morning cleansing.

Massage and electricity are recommended as adjuvants ; they are useful in atonic cases, but in stenosis they may prove positively injurious.

Drugs are of great value if used with discrimination. The best of these is creosote, which retards and sometimes prevents fermentation if given in doses of two or three minims in capsular form two hours after each meal. Papain with full doses of bicarbonate of soda may be given soon after a meal in all cases, especially where there is much organic acidity. It is a striking fact in the writer's experience that acids and pepsin are often useless, and sometimes injurious, even when there is a diminution of the free acid in the gastric contents. The dilatation caused by pyloric stenosis is usually progressive and ultimately permanent ; and though much relief follows lavage, it is in no sense curative.

Operative interference is therefore indicated in all cases where the obstruction is severe, and unquestionably the easiest, the least dangerous, and the one which gives the best results is gastro-enterostomy. Resection, pyloroplasty, and mechanical dilatation of the pylorus are often found most difficult and prolonged operations ; they are moreover liable to lead to relapse, and in some of the writer's cases gastro-enterostomy had to be performed afterwards.

This operation can be performed rapidly in all cases; and the writer, after witnessing the results in a considerable number of instances of pyloric obstruction, both benign and malignant, does not hesitate always to recommend it to the patient. When carried out skilfully there is no justification for any attempt at a reduction of the size of the dilated organ by further operative procedures.

The treatment of gastric tetany is still to be discovered. Though the condition is almost certainly due to auto-intoxication by products formed in the stomach, and the rational indication is the speedy removal of these, nevertheless death

has so often followed the act of introducing the stomach-tube that this procedure should not be resorted to in the presence of an actual attack of fully developed tetany.

Copious saline injections by the hypodermic and rectal routes, and a free diaphoresis by a hot-blanket bath, may aid elimination. Should the patient be able to swallow large draughts of water with the view of washing out his stomach without the aid of the tube, he certainly should be encouraged to try the plan.

GASTRIC CANCER

Etiology.—Cancer occurs more frequently in the stomach than in any other organ, though some authorities give the uterus as the most common site of the disease. From a study of very full statistics, it appears that primary carcinoma of the stomach falls little short of equalling all the other sites of cancer added together.

Males are more frequently affected than females, but not markedly so ; relative frequency diminishes with age, so that in subjects between sixty and seventy both sexes are nearly equally liable, women perhaps being in the majority. It is a disease of the post middle-age of life, being rare before thirty-five and most common between fifty and sixty. Seldom seen in the tropics, it is commonest, according to Häberlin, in Switzerland.

Heredity plays but a minor part in the incidence of the disease. In a small percentage of cases a previous definite history of ulcer may be found, but as a rule the victims of gastric cancer have previously been healthy individuals with excellent digestive powers. We know nothing more about the cause of cancer of the stomach than of the etiology of the disease when it attacks other regions. Trauma and infection have their advocates; but when we consider the abuse to which the stomach is so often subjected, and its exposure to infective organisms of every kind, these agents can have but a trivial predisposing influence. It occurs as frequently amongst the poor and under-fed as amongst the class who habitually over-load their stomachs with rich foods. Prolonged worry and grief certainly appear to act as powerful predisposing causes. A family history of tuberculosis is obtainable in a fair proportion of cases, but alcoholism is a doubtful predisposing agent.

Pathological Anatomy.—The pylorus is affected about as frequently as all the other portions of the stomach taken collectively ; next in frequency come the smaller curvature and the cardiac end ; only in a small percentage of cases are the anterior and posterior walls involved, and in a still smaller number the great curvature.

The most frequent form of gastric cancer is the spheroidal carcinoma, only about 30 per cent. of the cases being of the cylindrical type; both forms may be either of the *scirrhous* or *medullary* variety. In the former there is great increase of fibrous stroma with few blood-vessels, producing a hard slowly growing tumour which has little tendency to infiltrate neighbouring organs or to ulcerate, but which by the subsequent contraction of the fibrous tissue is very liable to cause stenosis of the pylorus. In some cases, where the growth involves the entire organ, its cavity becomes much diminished in size, forming the water-bottle stomach, which, though described as cirrhosis of the stomach, is believed by most authorities to be always of a scirrhous nature.

The medullary variety, consisting mainly of spheroidal or cylindrical cells and cell-nests and small amount of stroma, grows more rapidly, involving all the coats, contains many vessels, and is more likely to disintegrate and end in ulceration and metastases in the liver and peritoneum.

Colloid degeneration may also occur in either the cylindrical or spheroidal forms; but it is much more commonly met with in tumours of the spheroidal type, though upon the whole colloid or mucoid cancer of the stomach is rare. It forms large nodular tumours which rapidly involve all the gastric coats and extend to neighbouring organs. It may appear as a diffuse infiltration of the stomach-walls, which, on section, to the naked eye presents the gelatinous translucent material occupying large alveolar spaces.

Often one variety is associated with another, and transitional forms are constantly met with. Sarcoma of the stomach is rare.

The cancerous growth starts in the glandular epithelial elements of the mucosa, extends downwards to the submucous tissue, and steadily invades all the coats, finally involving the peritoneum, lymphatic glands of the abdomen, and often the liver, pancreas, and intestines; whilst secondary growths often occur in the cervical and inguinal glands, the pleura, lung, abdominal wall, and occasionally in the membranes of the brain or other distant parts.

Cancer of the stomach is primary in the great majority of cases, but the organ may become secondarily infected from neighbouring regions; and the pancreas, when the site of primary cancer, not infrequently is the origin of secondary gastric growths.

As the tumour increases in size, the portion protruding into the gastric cavity becomes liable to ulceration, and the ragged sore with its hard infiltrated edges is characteristic. Occasionally the disintegration of the growth extends deeply, and perforation into the peritoneal cavity may occur, though as a rule this is prevented by adhesions.

Of great importance is the condition of the glandular elements over some areas of the stomach. These are often found to have entirely disappeared, especially in the pyloric region; whilst sometimes the secondary atrophy of the peptic glands may be noticed all over the mucosa, chiefly involving the destruction of the parietal cells, even in cases where there is little evidence of interstitial infiltration. The capacity of the stomach may be greatly increased when the growth is situated at the pylorus so as to produce stenosis, but as a rule the colossal dilatations met with in benign stenosis are rarely approached in cancer. In disease of the cardiac end the organ is diminished usually in size and its coats obviously withered; but in the diffuse scirrhous variety of cancer, as already mentioned, the stomach-walls are very greatly thickened, and its capacity reduced to a fraction of the normal dimensions. Hour-glass contraction may occur in rare instances. The position of the diseased organ in the abdomen varies within wide limits; if not bound down by adhesions to neighbouring organs it may form a freely mobile tumour which descends towards the pelvis, dragged down by the weight of the neoplasm.

Symptoms. — There is difficulty in giving any general outline of the symptomatology of gastric cancer which will be applicable to more than a moderate percentage of the cases met with in practice, since the clinical picture varies so much with the site and dimensions of the growth, and whether or not ulceration or dilatation already has taken place. Nevertheless many symptoms are common to nearly all cases independent of localisation or secondary changes in the new-growth, and these will be first mentioned. Occasionally, however, these symptoms may be latent or may only appear towards the termination of the disease.

The mode of onset is usually insidious. Vague symptoms of dyspepsia are complained of; there is a feeling of weight and discomfort in the stomach, as if the food remained in it too long; belching of gas and often acid eructations are common.

Loss of appetite even at this early stage may be well marked; it is a symptom of much importance, and with it is often associated nausea. These may be so prominent as to overshadow other signs and symptoms, and may remain all through the course of the disease, the sight of food sometimes inducing loathing. In rare cases the appetite is unimpaired for a long time.

Pain soon comes to be a constant feature in most cases; it is of a dull gnawing or wearing character at first, but eventually may become as severe as the pain met with in ulcer. That produced by the growth is of the continuous type, but it becomes aggravated by the associated dyspepsia, so that some intelligent patients volunteer a description of the

separate pains felt during fasting and after eating. The aggravation of the distress does not occur so soon after food as in ulcer, and the pain is more diffused, being felt over a considerable area of the epigastrium, whence it may radiate to the interscapular region or pass backwards to the lower dorsal spines. When the growth ulcerates, the nature of the pain may change, coming on in severe paroxysms immediately after eating.

Tenderness is always marked over the epigastrium, often diffused like the pain, but it sometimes is found circumscribed and limited to the region of the pyloric tumour when this is to be felt. Various tender points may be made out in the skin from the level of the nipple to the umbilicus in front, and behind from the fifth to the last dorsal vertebra.

Anæmia, failure of strength, and *loss of weight* appear early and steadily progress, eventuating in the well-known and easily recognised pallid or sallow cancerous cachexia. The anæmia may be so profound as to simulate the pernicious type, and a not inconsiderable number of cases of gastric cancer are mistaken for a primary anæmia ; but it is rare for the red corpuscles to fall below half the normal count, though there may be considerable increase in the white cells and loss of hæmoglobin ; and unless the bloodlessness is caused by latent hæmorrhages at an early stage of the disease the emaciation is in proportion to it. Where there is retarded absorption of water the concentration of the blood may cause a great relative increase in the number of red cells.

The emaciation is progressive, like the other symptoms, and always becomes extreme. Here, again, mistakes are liable to occur in the interpretation of the symptoms. The wasting caused by the toxic influence of the neoplasm upon the general nutrition of the body is invariably progressive, but the emaciation which results from the anorexia and impeded gastric digestion is liable to variations, which may tempt the physician to alter his diagnosis.

One of the most commonly observed features in the progress of the malady in *hospital* patients is a slight transitory gain in weight following their admission, owing to the change of food, method of feeding, relief to the vomiting, &c., and it is always most important to remember that a moderate temporary increase in weight under such circumstances is not incompatible with malignancy.

Lavage may cause considerable temporary improvement in the wasting, but it is not so easy to explain the remarkable increase in weight which the writer has noticed in at least half a dozen cases of gastric cancer after a simple exploratory incision had been made for diagnostic purposes.

As a rule, the emaciation is not so rapid and extreme as that often observed in hepatic cancer and in some cases of diabetes ;

but a loss of almost 100 lbs. has been recorded in cancer of the cardiac region of the stomach, where the œsophagus had been blocked in a previously obese subject.

Fever is often present at some period of the malady's progress. According to Osler, it is noticeable in about half the cases, though Riegel states that febrile temperatures are very rarely observed during the course of the disease. Its intermittent character is probably the cause of its being often overlooked in cases occurring in private practice, as in the writer's experience febrile temperatures are noticeable in the majority of hospital cases at some stage of their progress, but the pyrexia is always of a fleeting irregular type.

Vomiting occurs in the great majority of all cases, but it is unusual for it to appear at an early stage. It is often absent when the disease is confined to the curvatures or posterior wall. It is constantly present in pyloric cancer, and does not occur for a considerable period after eating, often only happening once daily, generally in the evening. At a later stage, when stenosis has taken place, the vomiting has all the characteristics of that already described under 'Gastrectasis' (p. 447) the vomitus being large in amount, sour-smelling, and containing portions of food ingested several days previously.

When the cardiac end of the stomach is involved, vomiting comes on soon after eating; the vomitus contains much œsophageal mucus mixed with the unaltered regurgitated food.

In some cases the vomiting is frequent and distressing, apparently the result of gastritis. It is in the main true that in gastric cancer of all kinds the ejection of the stomach-contents affords little relief to the pain and discomfort of the patient. The chemical characters of the vomitus will be referred to later on.

Hæmatemesis occurs in a large percentage of the cases, though it is obvious only in less than half the number. It is usually slight in amount, appearing as coffee-ground vomitus; but when a large vessel is eroded in the necrotic process, a violent hæmorrhage may occur and prove fatal. The vomited matter, if repeatedly examined, will probably afford evidence of some degree of hæmorrhage in nearly every case at some stage of its progress, and it is often found in the lavage-washings when not visible in the ejecta.

Melæna is rare, unless there be no pyloric obstruction, for it is characteristic of pyloric cancer that the growth is limited by the pyloric ring and very rarely extends into the duodenum; but in the small round-celled lymphosarcoma which occurs in younger subjects such an extension often occurs. In cancer of the other portions of the stomach, where there is no pyloric stenosis, melæna is not uncommon.

Constipation is the rule, especially in pyloric cases, but occasionally diarrhœa occurs at intervals during the later stages. The tongue is furred, and a bad taste in the mouth is constantly complained of; the breath is frequently foul, and the eructations often offensive.

Urinary changes are not important. The secretion is scanty, and albumin, indican, peptones, and traces of acetone and sugar are often found. Œdema of the legs, beginning in the feet and extending upwards, only occurs towards the termination of the disease; it may be of renal origin or symptomatic of anæmia, or it may be associated with ascites caused by portal obstruction, when usually some jaundice will be present.

Physical examination of the abdomen is of greatest importance. The presence of a tumour may be demonstrated in the majority of cases as the disease advances towards its fatal termination; but in a not inconsiderable number of cases a tumour may be found as soon as the patient first falls under observation.

In the recumbent position, with the legs drawn up, the growth may be quite obvious to the naked eye; if not bound down by adhesions it descends on inspiration. It is most commonly situated in the pyloric region just outside the external border of the right rectus; and as it is always in close relation to the liver, it will be often seen to move up and down with this organ during breathing. If dilatation of the stomach has occurred, inspection will also often reveal its boundaries through the thin parietes, and on inflation, as described under 'Gastrectasis,' these become more evident. Moreover, inflation will often reveal to the eye a change in the position of the tumour, which usually will be found pushed towards the right side and somewhat depressed. A large tumour on the posterior surface may be visible, but it disappears as soon as the stomach is filled with gas.

If the stomach has fallen considerably downwards, tumours of the small curvature may be visible; and obvious peristaltic movements may be seen in pyloric stenosis, even when there is no evidence of a tumour upon inspection. Occasionally a small tumour on the anterior surface may be seen to apparently travel from left to right with the peristaltic wave.

Palpation gives valuable aid. The growth feels usually hard and often irregular or nodular; it is nearly always very tender. If situated in the small curvature it may be palpated only after causing the patient to take a deep inspiration. Where ptosis has occurred, palpation may reveal tumours situated in every region of the stomach, save at the cardiac end, and only the passage of a sound may reveal its suspected existence.

In the early stages of the disease most tumours are freely movable upon palpation; they afterwards tend to become

fixed by adhesions. The pyloric growths are as a rule very easily moved about to either side, upwards, or downwards. Where pulsation is marked, by changing the patient's position from the recumbent to the knee-elbow posture the weight of the growth is often removed from the abdominal aorta, and the pulsation disappears.

Percussion is most unreliable, and seldom affords any aid which is not available from palpation and inspection.

Examination of the gastric contents, after the use of the syphon or after spontaneous vomiting, affords invaluable information, and often by the syphon the diagnosis can be confidently made at a stage prior to the appearance of tumour, vomiting, or marked emaciation. The vomited matter, as already described, by its mere bulk and by the presence of undigested particles of food swallowed several days previously, betrays stenosis and dilatation in the later stages ; there are also numerous organisms, as sarcinæ and yeast, and one which is of most importance—the long thread-like bacillus first described by Boas and Oppler, which has the power of manufacturing lactic acid. Coffee-ground vomitus, or microscopic evidence of blood-cells, is highly suspicious. Occasionally fragments of the cancerous tissue have been detected in cases where necrosis has occurred.

The chemical examination is of primary importance, and should be repeated several times at varying periods after eating. Two results, though not strictly pathognomonic of cancer of the stomach, may be taken as almost conclusive, since they occur in such a very large proportion of all cases. These are the total absence or very marked diminution of free HCl and the presence of lactic acid. The method for demonstrating the free HCl has already been detailed upon page 337. The writer recommends for its convenience and rapidity the ·5 per cent. alcoholic solution of dimethyl-amido-azobenzol as used by Töpfer, and in case of doubt it may be checked by Günzburg's phloroglucin and vanillin reagent, or by the tropæolin test. The first-named liquid strikes a bright-red colour upon being added to the normal stomach-contents, even without previous filtration. Congo test-paper will turn from pale pink to blue under similar conditions.

If these reactions are not obtainable, it may be correctly assumed that free hydrochloric acid is absent and that the stomach is no longer able to digest food.

The test may be applied to the stomach-contents several hours after eating, but it is advisable to first wash out the stomach and let the patient take his ordinary food, and in six hours afterwards the syphon is again used, when the presence of coarse particles of undigested food will reveal loss of motor power, and the test will also demonstrate the absence of free HCl. A test breakfast may be given early in

the morning, such as that of Ewald and Boas, consisting of a couple of small rolls or a large slice of white bread and a cup of weak tea without milk or sugar, or a large tumblerful of water, and in an hour the contents of the stomach are syphoned off and tested. Riegel points out that after the test *breakfast* some free acid may be found, whilst six hours after a test *meal* of broth, beefsteak, bread, and potatoes, administered to the same patient, no trace of the acid may be visible.

Pepsin is usually markedly diminished or absent, as well as the acid, and both these results are easily explained by the glandular atrophy, which is always associated with the gastric cancer. There is usually diminution of free acid in gastritis, and in the old chronic cases it may be entirely absent; but the study of such cases shows that the disappearance of the acid only takes place after a long period of gastric distress, *whilst in cancer it is one of the earliest signs.*

Upon the other hand, it must always be remembered that free acid is found, sometimes even to excess, in a small percentage of cases of gastric cancer, and these cases are the few (5 or 6 per cent. at the most) where the carcinoma is grafted upon an old gastric ulcer, and they do not alter the general law that in gastric cancer there is a marked diminution in the acid-producing power of the gastric mucosa. Rarely is the acid production entirely arrested; though the free acid may be absent, some combined acid is usually present. The case mentioned upon page 339 may be here referred to as an illustration of the danger of founding a diagnosis upon the one sign of acid deficiency.

Lactic acid does not form in any appreciable quantity in the normal stomach, but in gastric cancer its presence is almost as constant as is the diminution of free HCl, and Boas maintains that the discovery of this acid in the stomach-contents enables the physician to diagnose cancer, often before there is any tumour to be felt. According to Riegel, two conditions are necessary for its production, and both these are present in gastric cancer—viz. a great reduction of gastric juice and a stagnation of the stomach-contents.

In chronic gastritis, with much atony, lactic acid may be present, but this is decidedly the exception; upon the other hand, however, it may be absent in cancer-cases of ulcerous origin, where there is hyperchlorhydria. The presence of the long thread-like bacilli which form lactic acid is also a valuable diagnostic point. In order to examine for lactic acid it is desirable that a special test meal should be administered which contains no lactic acid. Boas recommends that the stomach be washed out and a pint or more of thin oatmeal-gruel, made with a large spoonful of the meal or oaten flour and water, be administered; this should contain neither milk, butter,

nor sugar. In an hour or two the syphon is employed, and the contents tested by Uffelmann's freshly prepared reagent. This consists in a modified form of 2½ drs. of a 4 per cent. carbolic-acid solution diluted with twice as much water, to which half a drop of liquor ferri chloridi fort. is added; the resulting liquid is of a bright amethyst-blue colour, which strikes a canary-green colour with the filtered contents if the acid be present. Tests for the retarded absorption-power of the diseased organ are of secondary importance.

The course of the disease is always towards a fatal issue, and this may occur within a year of the first symptoms; as a rule it is later, but seldom exceeds two years, though several cases are on record where the duration has exceeded three years. In the prognosis of the duration it should be remembered that the hard scirrhous tumours are of much slower growth and less toxic in their effects than the softer growths which are also more prone to disintegration. Death has been known to follow as early as three or four months in some instances of these latter types of the disease.

Complications of various kinds may occur, some of which have already been incidentally mentioned, as perforation, hæmorrhage, adhesions, secondary growths, &c. Secondary growths are most common in the lymphatic glands; they may press upon the portal vein and cause ascites, or block the bile-duct and produce jaundice. Gastro-colic fistula may occur towards the end, and show itself by fæcal vomiting. Thrombosis of veins, as the saphena or femoral, is not rare, and sub-phrenic abscesses are sometimes met with. Gastric tetany and coma, described under 'Gastrectasis' and 'Diabetes,' are very rare events, but they do sometimes appear towards the end.

Diagnosis.—The chief points have been already emphasised in the foregoing description, but it must be always remembered that there is no pathognomonic sign or symptom of the disease, save in those rare cases where a fragment of cancerous tissue is discovered in the ejecta. Every case must be weighed upon its own evidence. The certainty of the diagnosis increases as one symptom or sign after another is added to the complex and variable clinical picture whilst the physician watches the development of the disease.

Often a positive opinion can only be given after patient watching and repeated examinations, especially when the disease is located in the cardiac end, and the growth cannot be palpated.

When the chief symptoms are all present in a given case there cannot be a mistake made in diagnosis. Thus, in an elderly, anæmic, emaciated patient, with gastric distress and vomiting of several months' duration, in which coffee-ground vomit is frequently or occasionally seen, and where the stomach-

contents constantly show absence of free HCl and presence of lactic acid, and where a tumour of the pylorus with gastric dilatation is present, the condition can be nothing else but that of carcinoma. If any one of these signs be absent, as pointed out by Riegel, there may be a difficulty; for example, all may be present save the characteristic chemical reactions of the stomach-contents, in which case the disease might be an ulcer which has caused stenosis and subsequent dilatation, with a palpable tumour, the result of inflammatory thickening.

Ulcer of the stomach occurs in younger subjects, chiefly in women in whom a previous history of former attacks is common. The pain follows food, or is aggravated by it after a shorter interval than in cancer. Appetite is usually good; there is little wasting as a rule, and often no anæmia; the vomiting affords marked relief and the blood brought up is usually bright in colour, and hyperchlorhydria is constantly present. If the symptoms have lasted for many months without marked cachexia, ulcer may safely be diagnosed. (See under the Diagnosis of 'Gastric Ulcer.')

Chronic gastritis gives a history of a dyspepsia of such long duration as to exclude cancer, in the great majority of cases by the time-limit alone. In some atrophic cases the symptoms of gastritis may be latent for years till motor insufficiency develops, forming a clinical picture impossible to differentiate from cancer unless the case is watched for many weeks or months after first coming under observation. There will be deficiency of free HCl in such a case, but never abundant lactic acid or thread-like bacilli or numerous blood-discs; a tumour is not palpable, nor is the cachexia well marked. As already mentioned, the absence of or deficiency in free acid is an early sign in cancer and a late sign in gastritis; and if the dyspeptic symptoms set in after the fiftieth year in a previously healthy patient there should be a strong suspicion of cancer in every case.

Anæmia of the pernicious type may closely resemble those cases of gastric cancer where the secondary anæmia is severe from the beginning. The blood-examination is useful. As already mentioned, the red cells seldom fall so low in cancer as in a primary anæmia, and the proportionate deficiency in hæmoglobin is less in anæmia; in other words, the colour-index is lower in cancer, each individual red disc being smaller, and containing therefore less hæmoglobin than in anæmia. (The presence of leucocytosis is of little value in the diagnosis of cancer of the stomach.) Moreover, in anæmia there may be little wasting, the free HCl may be normal, and there is no lactic acid. When a tumour is present the diagnosis is certain.

The different points in the localisation of the tumour in the stomach which differentiate gastric cancer from other

abdominal growths are already dealt with in the physical examination under Inspection, Palpation, Inflation, &c. The presence of foreign bodies in the stomach, especially the tumour which is caused by swallowing hair, can be differentiated by the history and by the absence of cachexia and other signs.

Treatment.—This is at the best but palliative, and consists of judicious dieting with easily digested foods, or with predigested milk or gruel. In most cases, owing to the repugnance to many foods, the physician will find it impossible to prescribe a hard-and-fast dietary, each patient's natural inclinations being often a more potent factor in the feeding problem than any scientific consideration.

Medicines are indicated for the relief of pain, for hæmorrhage, and for other prominent symptoms. As a rule, vegetable bitters are useless for the anorexia.

Lavage is of great value when pyloric obstruction is present; and when this is cautiously and gently carried out, vomiting, heartburn, and acid eructations are markedly relieved. Often creosote gives comfort by checking fermentation and diminishing pain, when administered in conjunction with the daily washing-out.

Operative surgery in gastric cancer has made marvellous strides during late years. Removal of pyloric growths in the early stage is followed, in suitable cases, by striking results; but often the operation in a given case has to be abandoned owing to difficulties caused by adhesions.

In the advanced stage of gastric cancer affecting the pylorus, or indeed all portions of the stomach save the cardiac end, but especially when dilatation of the organ is marked, surprising relief is afforded by gastro-enterostomy. The operation is as a rule easily performed, and has a slight mortality when performed by a surgeon who pays special attention to gastric surgery. Lavage is dispensed with, all vomiting, and most of the pain and distress and local symptoms, disappear, and the patient for a time usually gains considerably in weight. The food passes directly through the diseased and useless organ, and digestion is accomplished satisfactorily by the intestine. There cannot be a doubt of the considerable prolongation of the patient's life with a relative degree of comfort, even when the diseased mass is left untouched. The writer invariably lays this alternative before every patient, and those of them who accept it always express their satisfaction.

The rational operation is, however, obviously the one which removes the disease and surrounding area of the pylorus or gastric wall, and Mr. A. B. Mitchell has recently carried out with striking success a method of procedure which seems to be fruitful of further advances in dealing with gastric cancer. He makes an abdominal incision and rapidly performs a

posterior gastro-enterostomy, and at the same time a careful exploration of the tumour and of the surrounding organs is carried out. Should the growth be found to be one capable of removal, this is subsequently accomplished at the end of about ten to twenty days, when the intestinal and gastric junction has been established. In this way the fatal effects of one prolonged operation are avoided, and the patient has become, even in the short interval, much better able to bear the excision.

No objection need be considered against the sacrifice of the stomach-function entailed by the gastro-enterostomy, for already the glandular atrophy has destroyed the organ as a pepsin and acid forming machine.

In cancer of the cardiac end, where the lower opening of the œsophagus is involved, sometimes relief may be obtained by the cautious passage of a dilating sound, but it is a noteworthy fact that in both pyloric and cardiac stenosis sometimes a remarkable spontaneous diminution in the blocking occurs from disintegration or degeneration of the growth—probably only, however, in soft cancers. In some cases a Symond's tube can be inserted with marked benefit, as is done in œsophageal cancer.

GASTRIC NEUROSIS.

(See under DYSPEPSIA, pp. 333–344.)

GASTRIC ULCER

This affection is also known as Round Ulcer, Peptic Ulcer, Simple Ulcer, Perforating Ulcer, and also as Cruveilhier's disease.

Etiology.—The records of large numbers of *post-mortem* examinations show that Brinton was probably correct when he stated that one body in every twenty shows signs of recent gastric ulceration or of the cicatrices of former ulcers. Some authorities place the percentage at a much higher, and others at a much lower figure.

Sex.—Women are certainly much more frequently affected than men, probably more than twice as often, and possibly, as stated by Martin, three to five times oftener than men.

Age.—The disease is met with most frequently in women between the ages of twenty and thirty, and in men between the ages of thirty and forty-five. It is rare before the age of ten years, but it has been observed in even young children.

Occupation has some influence. Thus the disease is more common in young female domestic servants, especially in cooks, whose habit is to taste hot food; and the prevalence

of gastric ulcer in housemaids and cooks is much greater than can be explained by the relatively large number of this class of patient seeking hospital admission. Persons whose occupation exposes them to trauma or pressure over the upper part of the abdomen, as shoemakers, and those who are liable to swallow particles of glass, metal, or china, in factories where these substances are constantly in a state of coarse division in the air, are supposed to suffer more frequently than others. Tight-lacing is believed to act injuriously in a similar manner, but there is little evidence in support of this supposition.

Improper food predisposes to the disease, and it is upon the whole more frequent amongst the poor than the rich. In manufacturing cities, it is especially prevalent amongst the female operatives in factories and warerooms, who drink large quantities of strong stewed or boiled Indian tea at every meal, which, indeed, often consists of nothing else but tea. In Russia, where tea is very largely consumed by all classes, the disease is comparatively rare; but the Russian drinks his tea always in the form of a weak, freshly prepared infusion of the China leaf. The writer believes that the frequency of gastric ulcer in the large class of female operatives employed in the linen mills and factories of Belfast is explained by their habit of living almost entirely upon tea which is frequently consumed in the form of a hot, strong decoction, often several times re-heated, and accompanied by small quantities of bread. Domestic servants frequently fall into the same vicious habit, and, instead of regular meals, take, several times during the day, tea which is kept hot on the leaves by permitting the tea-pot to simmer upon the top of the kitchen range for considerable portions of the day.

Alcoholism does not seem to be a factor in the production of gastric ulcer.

Anæmia or *chlorosis* is so frequently found associated with gastric ulcer that the combination cannot be regarded as a coincidence, especially as hyperchlorhydria is a very constant feature in both chlorosis and ulcer. Whether the chlorosis should be regarded as the cause or effect of the stomach-complaint is not so easily determined as would at first sight appear. Perhaps the more rational explanation of the association would be found in the hypothesis that the chlorosis, hyperchlorhydria, constipation, and gastric ulcer are but different phases of the result of the same faulty hygiene.

Syphilis, tuberculosis, chronic renal and valvular diseases, and various septic conditions are supposed to bear some relation to the incidence of the disease. The influence of cutaneous burns will be referred to when detailing the etiology of duodenal ulceration. Heredity evidently, if at all to be regarded as a predisposing cause, plays but a very minor part.

Pathological Anatomy.—The form of lesion characteristic of gastric ulceration is the so-called peptic or round ulcer, which is only found at the lower extremity of the œsophagus, in the stomach, and in the proximal end of the duodenum—*i.e.* in those parts of the intestinal tract which come into contact with undiluted gastric juice. The sore is usually solitary; but two ulcers are not uncommon, and in a small percentage of cases several have been found, especially in acute types of the disease. The posterior surface of the stomach is the usual site, the lesser curvature and the pylorus suffering next in frequency; but if we define the localisation of gastric ulcer to be towards the pyloric region, on the posterior wall near to the small curvature, probably three-fourths of all cases will be included. The remaining fourth will cover the number of cases occurring upon the anterior wall, cardiac end, greater curvature, and fundus.

The size varies considerably; oftenest they measure from a half to one and a half inches across, but they have been found as large as the palm of the hand.

The edges are as sharply defined and clean-cut as if the ulcer had been punched out. In chronic cases the edges lose this sharp definition and become thickened, so that an indurated sinuous margin surrounds the sore. A recent ulcer is round or oval, and its floor may be the sub-mucosa, the muscular or serous layer, or it may have eroded all these, and the floor may consist of pancreas, liver, or other structure to which it has become adherent. As the necrotic action proceeds outwards from the mucous coat the opening in each successive layer becomes smaller, so that the ultimate shape is a funnel with the narrow part or neck at the peritoneal coat. This is sometimes spoken of as an ulcer with terraced walls. In old chronic cases the opposite condition may exist, and the thickened margins may be overhanging or undermined, but rarely are they rough or ragged in any case, either old or recent.

As healing occurs, there may be little or no scarring left when the ulcer does not extend beyond the mucous coat, but in deep and extensive sores the contraction of the newly formed scar-tissue leads to puckering and deformities, which may cause more or less complete stenosis of the pylorus, and consequent dilatation of the stomach, as described under 'Gastrectasis.' Where a large ulcer engirdles the stomach the subsequent puckering may produce an hour-glass contraction of the organ. In this condition the stomach is divided into two compartments—one in direct connection with the œsophageal opening, and the other leading into the pylorus; whilst the two spaces communicate with each other through a variably sized aperture in the middle. Whilst many ulcers heal spontaneously by the formation of granulation-tissue,

which springs from their margins, walls, and floor, sometimes, no doubt, without producing even any symptoms of their presence, there are others which extend deeply and widely, and which never heal.

The glandular elements participate in the healing process, but the cicatrisation destroys a considerable number of the tubules in the vicinity of the ulcer. Around the margins of open ulcers more or less evidence of inflammatory action is usually present, and in deep ulcers where adhesions have formed the inflammation may spread to the adherent organs. Localised peritonitis is common.

Perforation is liable to occur when the necrotic action is so rapid as to involve the serous coat before adhesions have formed ; hence the danger of ulcers on the anterior wall of the stomach, since adhesions do not usually occur in this situation owing to the motility of this portion of the organ.

When partial adhesions have formed to neighbouring organs the ulcer may still perforate into them ; in this way an abscess of the pancreas, liver, spleen, or pleura may be produced. Thus, in one case seen by the writer with Dr. S. B. Coates, a latent ulcer with partial adhesions to the liver perforated, the subsequent hepatic abscess involved the diaphragm and invaded the right pleura, causing a localised empyema which pointed between the ribs, discharging a large quantity of pus, and leaving a fistulous opening through which small fragments of parsley were seen to flow after a meal of chicken-soup had been administered some months after the perforation had occurred.

Localised pockets of pus may be formed between folds of intestine which have become adherent to the stomach, and large sub-phrenic abscesses are not rare, and these occasionally may contain air, constituting the condition known as sub-phrenic pyopneumothorax.

A pin-hole perforation may occur, and the rapidly formed adhesions which follow may shut out the extravasated stomach-contents from the general peritoneal cavity ; but as a rule the perforation is speedily followed by an acute septic peritonitis. The pericardium has been perforated, and a gastro-colic perforation ending in a fistula is not very rare. General emphysema has been observed in several cases to follow perforation.

Vessels are liable to be eroded in the necrotic process, and, especially in deep ulceration, large arteries may be perforated, like the splenic, coronary, or gastro-epiploic. The vessels in the vicinity of the ulcer are often found diseased, and sometimes the seat of thrombotic or embolic blocking, and occasionally miliary aneurisms have been seen.

Pathology. — The pathological problem, notwithstanding innumerable researches, still remains to be solved. Many of

the theories which have been brought forward to explain the pathogenesis of the affection are becoming only of historic interest as further light upon the digestive functions prove them to be untenable.

Pavy's speculation that the healthy stomach is prevented from digesting its own tissues by the alkaline state of the blood circulating through its mucosa has led to the theory that gastric ulcer is caused by some obstruction in a small vascular area in the stomach, which, being therefore not supplied by the stream of alkaline blood, becomes digested, and an ulcer results.

Independent of the auto-digestion theory and its relation to the alkalinity of the blood, there is a large amount of evidence accumulating which goes to prove that the first step in the morbid process is a shutting off of the blood-supply in some localised area. The name of Virchow is associated with the theory that embolism, thrombosis, aneurismal dilatation, or trophic changes in the vascular walls cause the primary blocking. Many other authorities have shown that such changes are commonly found in the vessels in the immediate area of a recently formed ulcer, and they infer that these are the primary cause of the digestion of the anæmic area, which has become robbed of its natural alkaline protection. Riegel is, however, probably correct in ignoring the latter consideration and regarding the primary blocking as a destroyer of the vitality of the part, which therefore by a deficiency of its vital resisting power becomes digested independently of any consideration of diminished alkalinity in the deeper layers of the mucosa, just as any piece of dead tissue introduced into the healthy stomach would be broken up by the gastric juice.

The next important question is, What is the *rôle* played by the hyperchlorhydria which is constantly found present in ulcer? It seems certain that this is a primary phenomenon, and not one caused by the presence of the ulcer. It probably is not the cause of the ulceration, as some suppose, but it is the main factor in preventing the healing process and, according to Riegel, it is the cause of the ulcer becoming chronic.

Sydney Martin, Letulle, and others have attributed to microbes the primary necrotic action which eventuates in the formation of the gastric ulcer.

Mayo Robson maintains that gastric ulcer is a mild form of sepsis leading to gastritis, and excess of free HCl in the gastric juice, and he believes that the source of the sepsis is usually oral. The writer has seen improvement so frequently follow the extraction of foul teeth and the disinfection of the oral cavity in ulcer of the stomach, that he insists upon this in every case as a preliminary measure in treatment.

Stockton's researches, strengthened by the experiments of Vedora and Yzeran, supply the basis of an explanation of the original blocking, or local anæmia. These latter authorities were able to produce changes in the mucosa, and sometimes typical gastric ulcers in animals, by injuring the splanchnics and vagus; and these ulcers differed from those produced by all other experimental methods in one important character which is highly suggestive. They showed little spontaneous tendency to healing, as is the case also in the human gastric ulcer. Hyperchlorhydria was a marked feature in the splanchnic cases. This is recognised as the *neuropathic* or *neurotrophic* theory of gastric ulceration, and may ultimately be found the best working hypothesis of the pathogenesis of the affection, and one which may be found capable of explaining the relation of chlorosis and cutaneous burns to peptic ulcers in the stomach and duodenum.

Symptoms. — In a considerable percentage of cases there are no indications or symptoms which suggest ulceration till a serious hæmatemesis or a fatal perforation occurs—in other words, the ulcer remains *latent.* The results of routine *post-mortem* examinations often demonstrate the cicatrices of ulcers which have never produced any evidence of their existence.

The disease usually is more or less gradual in the onset of its symptoms, which begin with some discomfort and weight or feeling of tenderness and distension, usually accompanied by acid eructations.

Pain.—This discomfort soon develops into a severe pain, which is the most characteristic and prominent symptom of gastric ulcer, and the one for the relief of which advice usually is sought. The nature of the pain, as described by ulcer-patients, is as varied as their traits of character: it may be said to be dull, boring, gnawing, stabbing, roasting, throbbing, burning, &c., so that little importance need be attached to the description of its mere quality, though sometimes intelligent patients will liken it to the suffering which they fancy would be produced by irritating an open sore—*i.e.* they describe it as of a distinct raw and pricking character,

Its chief features, however, are that it is usually circumscribed or confined to a limited area; it has close relationship to the time of eating, and to the nature of the food ingested; it often comes on in severe paroxysms, and it is promptly relieved by vomiting. First, as regards its site, this varies; but usually it is felt over some spot in the epigastrium, which is exquisitely tender to the touch, and in the same individual the localisation of the pain is always the same in the attacks. When the spasms are very severe, the pain may radiate from the tender spot all over the epigastrium, and beyond it; a very common condition is where the pain extends directly

backwards to the region of the eighth, ninth, and tenth dorsal spines. (Pain and tenderness must not be confounded with each other.) In a general sense only can the locality of the pain be interpreted as an indication of the site of the ulcer, but persistent dorsal pain is highly suggestive of an ulcer on the posterior surface of the stomach.

The relationship of the pain to the time of eating is highly characteristic. In the majority of cases it coincides with the ingestion of food ; rarely, however, does it appear immediately, but follows at a variable time in severe paroxysms, beginning as early as a quarter of an hour or as late as an hour, and occasionally not for two hours, after.

In a typical case the patient may sit down in total absence of pain at meal-time. A few morsels of solid food may induce a slight pricking pain in the epigastric region, which disappears during eating. In about half an hour afterwards, when the digestive process gets well started, a smart burning pain is experienced, which increases in intensity till a severe paroxysm develops, as abundant hyperacid gastric juice continues to be poured out, and there is no abatement or relief till vomiting brings up the gastric contents.

Often vomiting does not occur, in which case the pain fades away as the hyperacid chyme empties itself into the duodenum.

Continuous pain independent of food is exceptional ; but it is sometimes met with, and is then probably caused by adhesions or a localised peritonitis, or by the irritation of some neighbouring organ which forms the floor of a deep ulcer. It is often distinguished by the patient himself from the severe cardialgic paroxysm occurring at the height of the digestive process. Occasionally it may be produced by a continuous hyperchlorhydria, which occurs in fasting, since it is relieved by the bringing up of a small quantity of pure but highly acid gastric juice—a comparatively rare event. The continuous pain, when present, is always aggravated by food.

Sometimes the cardialgic paroxysm is stopped by eating some additional food—a phenomenon usually ascribed to the dilution of the gastric hyperacid secretion ; but the pain soon returns again. (This symptom will be referred to again in discussing the diagnosis of Duodenal Ulcer.)

As a rule, the length of the interval between feeding and the advent of pain cannot be taken as a reliable guide to the localisation of the ulcer, though it is generally stated to be much shorter when the ulcer is situated at the cardiac end of the stomach.

The nature of the food swallowed has a characteristic relation to the pain : this is always greater in intensity, and comes on more quickly, when hard or solid food is ingested, so that

some patients escape the paroxysms when living upon small quantities of bland liquids. It is often stated by patients that even a small draught of strong tea brings on a severe attack.

The posture assumed by the patient during the paroxysm is variable, but sometimes characteristic ; usually the recumbent position gives most relief, though some patients turn over on the face or bend forward or roll themselves upon the floor. If the pain be the result of adhesions a change of posture may entirely remove it, and in some such cases it only comes on when the stomach is distended by a large meal, where the ulcer has healed, but adhesions remain. As a rule, when this is the case, coarse food does not bring on an attack unless it be large in amount. Posture gives no reliable data for the localisation of the ulcer.

Some confusion exists in the minds of those authorities who have described the result of pressure during the paroxysm. As a rule, the issue is simple : a diffuse pressure like that produced by leaning over a pillow or water-bottle affords relief almost always, but a localised pressure by the hand or fingers, such as is made in a clinical examination, always aggravates the paroxysm when applied to any portion of the epigastrium. This detail is intimately associated with the next symptom to be described—viz. :

Tenderness, which, as already stated, must not be confounded with pain in gastric ulcer. It is a symptom often quite as characteristic as pain. Upon pressing with the tip of one finger over various spots in the epigastric region, a point which is markedly tender or painful to pressure may be discovered. This is oftenest in the middle line below the ensiform. The important character of this symptom lies in the smallness of the tender area, which may not be larger than the finger-tip. It is situated at a lower level in women, according to Brinton. A corresponding area may usually be discovered behind, about the level of the ninth dorsal vertebral spine. The localisation of the ulcer usually does not correspond with these tender points. The patient is sometimes unaware of the tender spot, though she knows that she cannot generally tolerate her corsets to be fastened.

Vomiting is often present, but it may be absent throughout in some patients who have seldom vomited during their ordinary good health even after eating irritating food. It is less important than the paroxysmal pain, but when associated with the sign to be next mentioned (hæmatemesis) it is of great value in the diagnosis. It may come on soon after eating, but as a rule it occurs at the height of the gastralgia or cardialgia—*i.e.* one or two hours after food—and it generally relieves this distress promptly. The food is ejected with little change when the vomiting comes on very soon after eating,

but usually it is in an advanced stage of disintegration owing to the hyperactivity of the gastric juice.

The stomach-contents are constantly hyperacid from excess of HCl, and the material ejected sets the teeth on edge.

Like the pain, it is much aggravated by hard coarse food, and may be caused to disappear by placing the patient upon a liquid diet. In some cases, especially with large deep chronic ulcers, it may be frequent and uncontrollable. When dilatation has occurred from pyloric ulcerative puckering, the act of vomiting is long delayed, and the ejecta may present all the characters described under 'Gastrectasis.'

Hæmatemesis is present in the majority of cases at some period of the disease, though it may be so trifling as to be often overlooked. It occurs as an obvious sign in at least one-third of the cases, and if microscopic examination of the vomitus be periodically undertaken, red blood-discs will often be found in numbers where the presence of blood was not suspected. Its origin is usually from a small vessel eroded in the wall or floor of the ulcer. Often the blood is bright red, or dark with little alteration; but when retained for any length of time it presents the usual well-recognised coffee-ground appearance. The former variety of hæmatemesis is the more characteristic of ulcer. Usually both forms occur at alternating periods.

The extensive hæmatemesis which occurs from the rupture of a varicose vein at the lower end of the œsophagus in hepatic cirrhosis must not be mistaken for evidence of a gastric ulcer. It is dark purple or black, and often wells up in great quantities, sometimes without vomiting, and usually unassociated with gastric pain.

Rarely is a large vessel cut across causing such hæmorrhage as leads to a fatal issue. It is, however, a common circumstance to find patients in a profoundly anæmic condition from repeated small hæmorrhages which pass down the bowel and are unnoticed. This is very liable to occur in duodenal ulceration, and in gastric ulcer without vomiting, constituting the following symptom:

Melæna, which is the name given to the stools which contain blood in the black tarry form assumed after being altered by the intestinal juices; and attention should always be directed to the motions in cases of suspected ulcer. It may occur with or without hæmatemesis.

The other gastric symptoms vary much. As a rule, appetite is not impaired, though the patient is often afraid to eat because of the pain. The tongue is usually clean, red, and often irritable-looking, and constipation is the rule. Acid eructations are common; these are the direct result of the hyperchlorhydria, and are not due to the usual organic acids met with in other gastric conditions, unless in stenotic cases.

Emaciation is not common unless in very chronic cases, and the patient may present a healthy appearance unless there be repeated bleedings ; but often anæmia to a marked degree may exist in cases without hæmorrhage, especially in the chlorotic type of ulcer. Even when emaciation and anæmia are present together, the usual cachexia seen in cancer is absent.

Examination of the abdomen reveals little, save when stenosis has occurred, except the characteristic tender points already described. There is no palpable tumour save in a small percentage of cases, but where the ulcer is deep and chronic an inflammatory tumour with much hard induration or thickening may be felt in the region of the pylorus or upon the posterior wall when the floor of the ulcer is formed of inflamed pancreatic tissue. Such tumours are apt to be mistaken for cancerous growths, and not unfrequently in the writer's experience the differentiation was difficult or impossible even when the mass was between his fingers after the surgeon had made an exploratory incision.

Chemical examination of the stomach-contents should never be omitted in doubtful cases, and usually the vomited matter will suffice for this purpose if due patience be exercised in watching for it about an hour after a test meal. It is not desirable to pass the stomach-tube if it can be avoided. Riegel, who first thoroughly investigated the amount of HCl in ulcer, finds hyperchlorhydria to be present in the very great majority of all cases, and this may now be accepted as the rule.

Perforation.—This occurs in at least one case in every twenty, and in the great majority of them the ulcer is found to be situated upon the anterior surface of the stomach, where adhesions do not form to the same extent as on the posterior surface.

The perforation often occurs during some unusually severe muscular exertion ; the lifting of a heavy pot from off the fire is a very common cause of perforation in domestic servants, and the accident is more likely to occur when the stomach is full. In a case under the writer's care in hospital recently a fatal perforation occurred in a patient who had swallowed absolutely nothing for seven days, rectal feeding being rigidly insisted upon because of continuous hæmatemesis. The direction in which the perforation may occur, and the after-results where more or less extensive adhesions have already formed, have been mentioned upon page 468. When the rupture is into the general peritoneal sac the stomach-contents escape, and very definite symptoms of severe general peritonitis rapidly supervene, with intense pain and often severe collapse. The abdomen becomes much distended with gas

and exquisitely tender. The shock produces a thready pulse, which sometimes cannot be felt; the features become pinched, the extremities icy cold, and a profuse cold sweat covers the cutaneous surface. Vomiting so rarely occurs that its absence, according to Traube, is diagnostic, though it occasionally occurs in pin-hole openings.

Examination of the abdomen reveals absence of hepatic and splenic dulness, but the writer attaches great importance to the quality of the note elicited by *gentle* percussion over the region of the stomach; the gastric quality is nearly always absent, being replaced by the hollow tympanitic sound caused by the general inflation of the abdominal cavity. By close attention to this point a perforation of the stomach may usually be differentiated from other forms of perforation, duodenal, intestinal, &c. But a pin-hole perforation near the larger curvature may cause severe symptoms, with the evidence of gas in the stomach. The writer has satisfied himself of the value of this diagnostic point in several instances where he percussed the abdomen immediately before laparotomy, undertaken for perforative peritonitis. A large distension of the cavity by gas escaping from a tubercular ulcer in the colon may push the stomach up into the thorax under the elevated diaphragm, where it may be discovered by a percussion-note very different from that obtainable over the abdominal walls.

Death usually follows perforation into the smaller or general peritoneal sac, in a number of hours if speedy surgical aid be not forthcoming. But occasionally spontaneous recovery results. The writer was fortunate in seeing this in three cases, but such a result must never be counted upon; it is only likely to happen if the stomach has been quite empty at the time of erosion or rupture.

Under 'Gastric Cancer' attention is directed to the fact that a small proportion of carcinomatous tumours take their origin in a chronic ulcer.

As already obvious from the general description of the symptoms of gastric ulcer, there are wide variations in the clinical picture. This has led to the acceptance of a number of distinct clinical types of the affection, which, however, need to be only briefly mentioned, as the individual symptoms have been already discussed.

1. The *acute perforating* ulcer. This is often, as already stated, entirely latent till perforation occurs; the ulcer is nearly always on the anterior wall, and is most commonly met with in young chlorotic subjects.

2. The *hæmorrhagic* ulcer. This, like the former, may be entirely latent till severe hæmorrhage appears; and as perforation may occur in an old intractable gastric ulcer, so severe hæmorrhage may also supervene in like manner in the most

chronic cases, and it may persist for long periods, as in the type styled the *scorbutic* ulcer by Lebert.

3. The *dyspeptic* form is characterised by symptoms not unlike those found in chronic gastritis, and there is absence of the typical, almost pathognomonic, attacks of pain occurring during digestion, or else these occur at irregular intervals, and hæmorrhages are usually entirely absent, but hyperchlorhydria is usually a constant feature.

4. The *gastralgic* or *cardialgic* type of ulcer shows the paroxysmal attacks of characteristic pain in their most distinctive form, as already described. In this form the resemblance to a gastric neurosis is close.

5. The *vomitive* form is sometimes met with in its typical condition, where the constant vomiting after every meal masks or overshadows the other symptoms.

6. The *recurrent* form is often met with, and may be an example of any of the above types, in which the symptoms entirely disappear after apparent healing of the ulcer, only, however, to return after certain long intervals and run through the same course again. Either fresh ulcers form in these cases, or the old sore has never completely healed, or the cicatrix may break down. Many of these ulcers pass into the form which might well be styled the *chronic* and *incurable* type.

7. The *cachective* form, as described by Lebert, is usually seen in the late stages of any of the above types. Its main clinical symptoms resemble those found in cancer of the stomach.

8. Mayo classifies all forms of gastric ulcers met with in operations under two heads—the *indurated* and the *non-indurated*, clinical, or medical ulcer, which only involves the mucosa and gives no evidence of its site till the organ is opened.

Prognosis.—The duration of gastric ulcer is most variable. It is manifestly impossible to formulate any general statement about the probable duration of an affection which is so often entirely latent. Many ulcers evidently heal rapidly, whilst others never heal, but remain permanently incurable unless surgical interference be decided upon. The prognosis is, however, upon the whole favourable, where the history gives good grounds for the belief that the ulcer is of recent origin, and where the patient will submit to prolonged rest and rigid dieting.

When the symptoms are of long standing the outlook is gloomy, especially if there is evidence that the ulcer is deep, if chronic local peritonitis exists and extensive adhesions have formed, and if the hæmorrhages are frequent and copious. A high degree of hyperacidity and much hypersecretion are unfavourable to healing. The prognosis is

most serious in ulcers on the anterior wal'. and in the recurrent form of the disease. The prognosis of stenotic cases is that of gastrectasis.

The marked advances in gastric surgery of late years are rapidly reducing the fatality of the affection, and it is probable that the all-round mortality at present is under 5 per cent. in the hands of physicians who advise excision and other operative measures where recurrent hæmorrhages, adhesions, and exhaustive vomiting threaten to prove fatal.

Diagnosis.—If the typical paroxysmal pain be constantly present after food and varies in degree with the coarse or bland character of the food, if localised tenderness be present at a spot in the epigastrium and a corresponding point near to the spine, if hæmatemesis and hyperchlorhydria be also present, there cannot be a doubt about the diagnosis. It is, upon the whole, not often that all these signs are present together, and then the physician will have to weigh the value of those which are present against the negative evidence. Hæmorrhage is the most valuable single sign, but it is risky to diagnose ulcer when hæmorrhage only is present. It is, however, less pardonable to be confident of ulcer when pain or gastralgia is the one symptom present.

The blood vomited is mixed with the food, and though coming from the stomach it may have trickled into the pharynx from the nares and been swallowed, or it may have come from the lower respiratory tract and have been afterwards swallowed, or it may have its origin in a ruptured varicose œsophageal vein in hepatic cirrhosis, or it may be caused by a hyperæmia of the stomach produced by valvular disease, or it may be the bleeding from a malignant growth. If all these sources can be excluded, and with them the rare form of vicarious hæmatemesis seen in menstrual suppression, the diagnosis of ulcer is highly probable, and certain if the vomitus be hyperacid from free HCl.

Functional or *nervous gastralgia* or *cardialgia* closely simulates gastric ulcer in the absence of hæmorrhage; but other nervous phenomena will be also present, and the pain never, or seldom, bears the same close relationship with the time of eating and the nature of the food as is the rule in ulcer. In many cases of nervous gastralgia food relieves the pain, and this is very rare in ulcer. When the gastralgic attack passes off there are usually no dyspeptic symptoms in the nervous affection, and the intervals between the attacks are very variable. The appetite in ulcer is usually unimpaired, but in the functional affection it is capricious and sometimes depraved.

Pressure made with the fingers relieves the pain in nervous gastralgia, but aggravates it in ulcer, and there are not the characteristic tender points of ulcer to be found. Hyper-

chlorhydria is present in both affections, but it appears and disappears irregularly in nervous gastralgia.

Gastric cancer is often differentiated with difficulty. The following points may be enumerated, which contrast the two diseases: The age in cancer is usually about forty years and upwards; in ulcer from twenty to thirty. The time-limit is of great value, if we except the small percentage of cases of cancer which develop upon the ulcerous foundation; the malignant disease usually runs a fatal course within two years, and if a case presents itself where the symptoms are of a duration of three or more years cancer is highly improbable.

The tongue is red and abnormally clean in ulcer, and the appetite unimpaired; in cancer it is always coated, and the appetite is usually lost, and often there is marked aversion from or loathing to solid food. Pain is generally a minor symptom in cancer; in ulcer it is usually the most prominent feature, and has the characteristic relationship with the food. Vomiting, generally present in both, has distinctive features in each: in ulcer it comes on early—within a couple of hours—and affords speedy relief; in cancer it is much later, and fails to relieve the abiding nausea or continuous gastric discomfort.

The appearance of the vomit gives valuable information. In cancer the food is slightly digested: in ulcer, unless speedily rejected, it is already in an advanced state of disintegration or solution; in cancer it is large, as several meals are brought up together: in ulcer the bulk is small. In cancer the organic acids are easily recognised by their sour odour; in ulcer there is no time for organic acid fermentation. Blood may be present in both cases, but the presence of fresh unaltered blood is strongly in favour of ulcer.

Chemical examination gives the best evidence. In cancer there is marked deficiency or absence of HCl and pepsin; in ulcer, hyperchlorhydria is the almost constant condition.

Emaciation and loss of muscular power with the cachexia mark the malignant affection; whilst in ulcer these are absent, save in the late stages of very long-standing cases. It is often very striking to observe a florid healthy complexion in patients who have suffered for long periods from simple ulcer of the stomach.

Lastly, the presence or absence of a tumour in the gastric region must be considered; in ulcer, such is absent as a rule, whilst in cancer the opposite is the fact. In old ulcers, especially about the pylorus, inflammatory thickening with muscular hypertrophy may often produce a tumour, which cannot be differentiated from a malignant growth even when the mass is between the surgeon's fingers during an exploratory operation. In such a case, where the other symptoms are not distinctive, there is no other course but that of waiting and closely watching the growth; if it steadily increases under

observation during some weeks, a malignant affection is certainly present.

Gall-stones produce symptoms which have frequently been mistaken for gastric ulcer. The colicky paroxysm produced by a gall-stone comes on usually with great suddenness and great severity, and persists for an indefinite period, independent of the digestive function ; the intervals between the attacks are markedly irregular. The pain, often amounting to agony, is more severe than in ulcer ; it radiates from the gall-bladder region towards the right shoulder. The tender point is over the gall-bladder, which may be found enlarged. Jaundice often follows, and a smart febrile temperature is usually observed ; and in a considerable number of cases the liver-margin may be felt below the costal arch.

Duodenal ulceration may be here described briefly. Its pathology and pathological anatomy correspond to those of gastric ulcer; it is usually found in the first part of the duodenum, and a considerable number of cases are on record where the ulcer has formed during the early stages of the healing of extensive burns of the skin of the trunk or limbs. The symptoms are practically identical with those described in gastric cases. The following differential points should be noticed:

The pain is usually felt to the right and above the umbilicus ; it occurs at a later period than in ulcer. Hæmorrhage may come *viâ* the stomach, but melæna without hæmatemesis, if recurring often, is very characteristic ; the black tarry motions should be looked for in every case of ulcer where the pain is delayed for three hours after eating. The symptoms are very often latent till melæna appears, and vomiting is no part of them. The disease is said to be less common in women.

There is a valuable diagnostic sign which the writer has found frequently most useful. If three hours or more after an ordinary meal severe gastralgic pain commences, which is promptly stopped by the swallowing of a small hard crust or a small portion of a dense dry biscuit, the ulcer is probably duodenal. The pain of ulcer is often arrested by giving food, but only when the quantity of it is sufficient to dilute the hyperacid gastric juice. In the duodenal test, the amount of food which will sometimes give the above reaction is incapable of doing this, but it causes the pylorus to contract reflexly by the mechanical irritation which it produces; moreover, in gastric ulcer the hard fragments would probably increase the pain.

Treatment. — In the author's 'Dictionary of Treatment' (4th ed.), pp. 311–317, the details of the therapeutics of gastric ulcer are enumerated ; only the principles need be here referred to.

Absolute rest in the horizontal position in bed is essential, and the writer insists upon this being persevered in for four to six weeks, even knitting and sewing being prohibited ; if reading is permitted, only a light booklet should be allowed—a heavy volume requires for its support an amount of muscular exertion which is objectionable. Rest to the stomach should be as complete as possible, and in cases where pain and hæmorrhage are prominent rectal feeding alone should be resorted to for ten days or more.

For routine treatment, by far the best dietary is milk, but everything will depend upon the way in which it is administered.

By feeding the patient from a large spoon the liquid nourishment may be made to pass directly through the stomach into the duodenum without exciting reflex contraction of the pylorus in those cases where the organ is not over-sensitive, as will be referred to under ' Chronic Gastritis.' Since this method was impressed upon the writer by the late Sir Andrew Clark he has adopted it with marked success in the treatment of ulcer. The milk may be boiled, peptonised, or mixed with an alkaline water. The same principle of feeding may be applied to the administration of beef-soups and jellies and other foods. The only objection to this rigid method of dietary is that it often necessitates a sojourn in a well-regulated hospital.

Usually all the symptoms are speedily relieved, or disappear, and often a complete healing of the ulcer occurs in the time above specified. A return to ordinary diet should not be permitted for very many months ; bland and smooth farinaceous foods with strained vegetable and animal soups, or broth, are quite sufficient to maintain a good standard of nutrition for long periods.

Drugs are indicated for the relief of vomiting, hæmatemesis, acidity, constipation, and anæmia, but except for the latter two conditions the rest and feeding cure usually meet all requirements.

Where the patient refuses or is unable to rest, and must follow his ordinary occupation, there is no drug so useful as papain, given in doses of 3 or 4 grs. in combination with 30 grs. of bicarbonate of sodium and an equal amount of magnesia, with $\frac{1}{18}$ gr. morphia administered within half an hour after food, which should always be soft or liquid ; 2 or 3 mins. of pure creosote two or three hours afterwards in the capsular form relieve pain and flatulence and prevent vomiting. Bismuth is, in the writer's experience, a much overrated drug in the treatment of ulcer ; and Carlsbad salt and Carlsbad water possess no specific virtues, but are often valuable adjuncts to the rest-cure.

The only reliable drug in severe hæmatemesis is a drachm

dose of chloride of calcium, given by the rectum, dissolved in 5 or 6 oz. of water, alone or in conjunction with saline transfusion or subcutaneous injections. When, in spite of rigid dieting and rest, the symptoms continue, or when they return after the patient has resumed his ordinary avocation, or when hæmorrhage has recurred to an alarming extent, or where adhesions are causing distress, the question of surgical interference becomes a practical one. It may be safely affirmed that many lives which might be saved by the surgeon are lost by persisting in medical treatment. In all the conditions named an exploratory incision should be made, and the case dealt with according to the state of the stomach revealed by inspection. The mortality of the exploratory incision is not more than ·5 per cent., and in the great majority of cases the conditions are found to be such as fall within the category of the recently established legitimate operations on the stomach. Thus a bleeding or chronic ulcer can be excised, or its area infolded; adhesions can be divided or separated; hour-glass contractions can be completely remedied by gastroplasty or gastro-gastrostomy; and an otherwise deep incurable ulcer may be made to heal after a gastro-enterostomy, which diverts the food directly into the jejunum.

The resources of surgery were taxed in one grave case under the writer's care, where, after years of suffering from symptoms of recurring ulcer, he advised operation. Mr. A. B. Mitchell, on making an exploratory incision, found an hour-glass contraction and a chronic unhealed ulcer. Both conditions were successfully remedied by gastro-gastrostomy and excision of the ulcer, and complete relief followed for a period of one year, when symptoms of fresh ulceration returned, followed by perforation, which was successfully dealt with by another surgeon, who performed laparotomy and excised the newly formed ulcer. After a period of seven months life became once more unendurable from the pain caused by adhesions. Laparotomy was again performed, the adhesions removed, and a posterior gastro-enterostomy performed by Mr. Mitchell; marked relief from the severe dragging pain and distress has been maintained since. In this case the writer had the opportunity of inspecting and handling the stomach through the abdominal wound upon the three separate occasions.

Peptic ulcer of the jejunum has been repeatedly found as a sequela of gastro-enterostomy. Mayo Robson records sixteen collected cases, nearly all occurring after the *anterior* operation, and in all perforation occurred. He refers to 236 cases of posterior gastro-enterostomy in his own practice and that of a colleague in which there was no evidence of this sequela.

Perforation should always be met by opening the abdomen, suturing the perforation, and infolding the diseased area by a double line of sutures, as soon as the patient has recovered from the initial shock. Mitchell has performed the operation thirty times, with seven deaths, and has had nine consecutive successes.

In Crisp English's list of fifty consecutive laparotomies for acute perforations, forty-two were in the stomach, with twenty-two recoveries; eight were duodenal, with two recoveries; in thirteen of the twenty-one cases which came to necropsy other gastric ulcers were found, their most common situation being exactly opposite the perforated ulcer.

GASTRITIS

The groups of so-called inflammations of the stomach still remain in a state of confusion. The practice of labelling as 'gastritis' every case of gastric derangement which does not arise from ulcer, cancer, atonic or stenotic dilatation, or neurotic disturbance is not supported by pathological evidence. The well-marked group of symptoms formerly recognised as acute dyspepsia is now in most text-books to be found under the heading of 'Acute Gastritis' or 'Acute Gastric Catarrh.'

The older nomenclature seems preferable till several physiological difficulties in connection with digestion are cleared up. Hence under the title 'Dyspepsia' in the present volume are included the acute and chronic affections so frequently met with in practice. According to most modern authorities, the former variety of affection corresponds to a mild simple gastritis or an acute catarrhal inflammation of the gastric mucosa, and the latter type to a similar simple chronic inflammatory process.

ACUTE CATARRHAL GASTRITIS

The name may be accepted as a synonym also of Acute Gastric Catarrh.

Etiology. — The condition may be theoretically regarded as present after the operation of the etiological factors mentioned under 'Acute Dyspepsia' have produced a degree of inflammation which does not immediately pass away with the removal of the irritating causes. These are, as already detailed, improper food, food cooked badly, proper food imperfectly masticated or taken in abnormally large amounts, especially after long fasting, copious draughts of iced water or of very hot liquids, strong alcoholic solutions, &c. The condition in its acute form may be secondary to organic

disease of the stomach as seen in cases of ulcer, or it may arise during the progress of a typical chronic gastritis or be but a local manifestation of some primary constitutional disease, as often observed in the exanthematous and other fevers and numerous septic or infective disorders. In Bright's disease an acute catarrhal inflammation of the gastric mucosa is often observed, probably the result of a vicarious attempt at the elimination of urea or ammoniacal salts.

Pathology. — The condition is rarely fatal, and the pathological changes are consequently seldom observed; when death is due to gastritis it is usually in the toxic or in the phlegmonous forms. The anatomical changes observed are such as occur in the catarrhal inflammation of other mucous membranes; but there is seldom any evidence of active hyperæmia, as this is known to pass away soon after death. It is, however, occasionally met with during operations undertaken for the relief of organic gastric affections in which sometimes evidences of acute catarrhal inflammation may be seen in the living subject. The membrane is swollen and appears somewhat softened in consistence, either about the pyloric end or in patches throughout, rarely as a general opaque cloudy swelling uniformly affecting the entire lining of the organ. Where the inflammatory action is best marked small hæmorrhagic extravasations and minute abrasions may be found, but these are always superficial. The epithelial elements in the gastric glands are swollen, and many of them are seen undergoing granular and fatty degeneration. There is marked increase in the goblet or mucus-secreting cells, and the entire thickness of the mucous coat may be found infiltrated with leucocytes, and always large amounts of free glairy mucus of a tenacious or ropy consistence are to be found adherent to the folds of the mucous membrane. When the condition has been produced by septic organisms or putrefactive bacteria in the food, such may be found in the stomach; but as a rule the substance which originated the catarrhal inflammation has been long since expelled by the vomiting.

Symptoms. —These consist of the pain, tenderness, nausea, vomiting, &c., which go to make the symptom-complex of acute dyspepsia, and, as in that condition, the vomit shows the food very little altered in appearance after its longer or shorter stay in the gastric cavity. The explanation of this appearance is due to the fact that the true digestive function of the organ is in complete abeyance, little free hydrochloric acid and pepsin being manufactured, though tough mucus is produced in large quantities. In a typical acute attack of dyspepsia all the symptoms rapidly disappear after the ejection of the exciting cause; but when the irritation has been severe there may be a prolonged anorexia, nausea, retching.

or continuous vomiting, accompanied by gastric tenderness and more or less persistent pain over the epigastrium. The persistence of these symptoms may be accepted as evidence that a catarrhal inflammation of the gastric mucous membrane has set in. The tongue becomes small, red, and irritable much more frequently than large and furred ; thirst is marked, and the vomiting may be so severe that even small draughts of iced water are immediately rejected. At a later stage organic acidity and other forms of distress will appear. There may be a considerable degree of collapse follow the continuous efforts at vomiting, the pulse becoming quick, weak, and thready, the surface of the body covered with a cold moist sweat, and the urine diminished in amount.

The duration of the affection depends largely upon the severity of the initial irritation ; it may be indefinitely prolonged if the etiological factor be not eliminated from the food. Thus the condition in alcoholics may remain as long as alcohol is continued, or it may become sub-acute or pass into chronic gastritis. When the condition supervenes upon a state of the mucous membrane like that existing in chronic valvular disease or renal affections, the vomiting may prove uncontrollable.

Treatment. — This is to be carried out upon the lines discussed under ' Dyspepsia.' After vomiting has set in the exciting agent has already been ejected, and emetics are dangerous. Small quantities of iced water or iced milk may be cautiously administered by the mouth, and rectal feeding introduced in all severe cases. As a rule, the best internal remedy is the carbonate of bismuth, made up with fresh acacia-mucilage combined with not more than $\frac{1}{25}$ gr. morphia and 2 or 3 mins. of dilute hydrocyanic acid. Counter-irritation to the epigastrium and flushing-out of the colon periodically are beneficial.

Special forms or varieties of inflammatory action in the gastric mucosa are, however, occasionally met with which should not be confused with the symptom-complex usually recognised by the title ' dyspepsia.' These are :

ACUTE TOXIC GASTRITIS

The term is a relative one. Most of the agents enumerated upon page 328 might be strictly regarded as toxic, but the term is used by most modern writers as indicative of a severe acute inflammation of the mucous surface of the stomach, produced by various poisons, as the mineral acids, alkalies, arsenic, phosphorus, and mercuric salts, in concentrated form. The symptomatology, pathological anatomy, and diagnosis and treatment of this form of gastritis belong to the domain of toxicology.

ACUTE SUPPURATIVE OR PHLEGMONOUS GASTRITIS

This is a rare disease, and occurs in two forms, in one of which a diffuse interstitial suppurative inflammation invades a large area of the stomach, whilst in the other variety the inflammatory action is more or less circumscribed, and ends in one or more gastric abscesses.

The disease usually commences in the sub-mucous layer, and it may be confined to this stratum, which ultimately becomes converted into pus ; but all the coats of the stomach are usually more or less involved and greatly thickened, the muscular layer suffering always least.

The peritoneum, when involved, may perforate in both varieties before death occurs.

The pathology of the affection is probably a streptococcic infection, since streptococci have been found in several instances in the sub-mucous layer.

In the majority of the cases the disease is primary or idiopathic, no focus of infection being apparent, but several cases have been found associated with septic puerperal conditions and pyæmia ; possibly infective endocarditis may have been overlooked in some cases. Cancer of the stomach and trauma have been noticed as the starting-point in a percentage of reports of the disease, and ulcer in only one case.

The condition brought about by severe bouts of drunkenness seems to prepare a way for the entrance of the infective organisms, which may be swallowed with the food or reach the mucosa from the blood. The disease is much more frequent in males, and the majority of the cases (not more than one hundred have been reported altogether) occur about middle-life or soon afterwards.

Symptoms. — The disease sets in suddenly with severe gastric pain and bilious vomiting, and some fever. The pain, at first confined to the stomach-region, spreads over the entire abdomen when peritonitis supervenes, which is in about half the cases. Before the advent of peritonitis there are great gastric tenderness and increased resistance on pressure, and the abdomen is distended. Constipation is the rule, but diarrhœa has been observed. The constitutional symptoms soon become severe, the pulse early getting quick, weak, and irregular ; and restlessness, delirium, and stupor supervene before death occurs, which may be in three or four days, though occasionally in the circumscribed form it may be delayed for several weeks. It is a remarkable fact that pus has not been found in the vomited matter except in a few cases of the circumscribed form where a localised abscess had formed.

The cases bear a strong resemblance to severe toxic gastritis, but the absence of a history of the administration of a

concentrated inorganic poison, and of any evidence of corrosion in the mouth, is usually sufficient to exclude this. The majority of the cases are not capable of being diagnosed during life.

The prognosis is always a fatal one, all treatment being unavailing.

MEMBRANOUS OR DIPHTHERITIC GASTRITIS

Is a rare condition found as a complication in diphtheria, scarlatina, and measles in children, and in small-pox, typhoid fever, diphtheria, ulcerative endocarditis, Bright's disease, and tubercle in adults. There is usually membrane visible in the throat, and the diphtheritic bacillus has been demonstrated in membranous patches found in the stomach. Most authorities believe that there is a croupous variety of the disease of the same nature as membranous enteritis.

Membranous or croupous patches are not uncommonly found adherent to the injured gastric mucosa in cases of toxic gastritis. *Parasitic* gastritis has been described as occurring as a very rare event in favus, in thrush, and after the introduction into the stomach of maggots or the larvæ of flies and beetles.

CHRONIC GASTRITIS

As already stated, this term is usually employed as a synonym for chronic dyspepsia. The symptoms have already been described under 'Dyspepsia,' on page 330.

When the causes which have led to arrest of or interference with the digestive process have been of very long duration or of unusual severity, certain well-marked changes are found in the gastric mucosa, so that long after these causes have been removed a condition of chronic inflammation may remain which produces more or less well-marked clinical symptoms, constituting a disease with the following definite anatomical or pathological basis.

Pathological Anatomy.—The gastric membrane is swollen, thickened, and covered over with a layer of tenacious mucus. The changes are not, however, confined to those usually associated with the use of the term 'catarrhal inflammation.' The process extends much beyond the surface, so that the microscopic and macroscopic examination reveals degenerative and inflammatory processes involving the parenchyma and the interstitial tissue of the organ.

The pyloric region is most deeply involved. The entire mucous surface is usually of a grey colour and is considerably increased in thickness. This increase in thickness is caused by a proliferation of the intertubular tissue with small-celled growth which causes by further pressure injury to the peptic or secreting cells already damaged by parenchymatous

inflammation. As it extends downwards into the sub-mucosa it may cause the mucous membrane to project in irregular polypoid excrescences.

As the inflammatory process proceeds the final result is wasting or atrophy of the mucosa, and almost complete destruction of the secreting cells of the tubules by mucoid transformation. In advanced cases the glandular tissue may entirely disappear, though there is usually considerable hypertrophy of the muscular coats, especially in the pyloric region.

A *sclerotic* condition of the stomach is sometimes seen in which its walls are much thickened and the cavity greatly diminished, constituting the so-called *cirrhosis ventriculi* of Brinton. This fibroid change is supposed by some to be an advanced stage of the chronic inflammation which commenced in the mucosa; by others it is held to be a hypertrophic sclerosis starting in the peritoneal coat and progressing inwards.

Symptoms.—Under 'Chronic Dyspepsia' the symptoms of disturbed digestion have been already described, which are met with in the earlier stages of the affection. There is pain and gastric discomfort of varying degrees of intensity, with flatulent eructations and occasional vomiting, but these are often transient or only appear after certain articles of food have been indulged in. When the inflammatory changes above described have become established, the symptoms tend towards the *continuous* type. The tongue is usually coated, the breath is foul; the appetite is usually very much diminished or entirely absent; a bad taste is constantly present in the mouth, and nausea is a prominent feature, especially in the mornings before eating, but it may only occur when the stomach is full; often it is present at all times.

Vomiting is not so marked a characteristic as nausea or anorexia; when it occurs valuable information may be obtained by a careful examination of the ejecta. The morning vomit contains little but slimy mucus, often tinged with bile; during the day the act of vomiting some hours after eating brings up the food unchanged, and each particle is coated over with this thick, tough, slimy mucus. The presence of such mucus intimately mixed up with the food is almost characteristic of chronic gastritis.

In advanced cases, where the motor power of the stomach is much impaired, there will be evidence of fermentation with great excess of organic acids; this is associated with acid eructations, pyrosis, gastralgia, and the sensory symptoms described under 'Chronic Dyspepsia,' and should be taken as suggestive of dilatation.

There is tenderness diffused over the entire stomach-region; and if dilatation has already occurred, which is not often,

inspection and other methods will reveal the enlarged organ; usually the stomach-area is normal.

Tests as regards the rate of absorption are often fallacious, as in many cases, owing to muscular hypertrophy, the liquid contents are quickly driven through the pylorus. The use of the stomach-tube settles the diagnosis in most cases easily. The two main characteristics are nearly always present in the matter drawn off—*i.e.* there is abundant adhesive mucus mixed intimately with the fragments of the food, and there is diminution of gastric juice, and nearly always marked deficiency of free hydrochloric acid.

Boas and others have described a form of chronic inflammation of the stomach as *gastritis acida*, but Riegel is satisfied that such is rare. The continual absence of free hydrochloric acid where cancer is out of the question may be taken as very strong evidence of established gastritis.

When the disease has produced marked gastric atrophy—the so-called '*atrophic gastritis*' of Ewald, which is but the final stage of chronic gastritis—there is total absence of free hydrochloric acid, pepsin, and rennet-ferment; this condition corresponds to *achylia gastrica*. It is, however, found in diseased conditions like pernicious anæmia, where there is grave atrophic change without symptoms of previous gastritis, and it is believed to be in some other cases a neurosis without atrophic change.

Even in advanced chronic gastritis the mucus may still be abundant. Schmidt has shown that a new form of epithelium appears in the stomach resembling that found in the intestines; it grows over the atrophic portions of the mucosa and secretes mucus freely.

The bowels are usually constipated, and nervous symptoms such as headache, insomnia, depression of spirits, and incapacity for work are common, and vertigo is often noticed.

When atrophy has occurred, and especially where the motor power of the stomach is much impaired, the general nutrition of the body suffers and marked emaciation often ensues, so that the clinical picture may closely resemble cancer.

Diagnosis.—The points have been sufficiently dwelt upon which distinguish chronic gastritis from other stomach-affections. The milder transient phases of the commencing disease described under 'Dyspepsia' are recognised by the history of their causation. As insisted upon by Riegel, a diagnosis of the established disease should never be attempted without repeated examinations of the gastric contents obtained after fasting and at variable periods during the digestive process.

Carcinoma of the stomach is the condition most likely to cause difficulty, but usually it can be differentiated by the history and chemical testing of the gastric contents. It is only

in long-standing cases of gastritis that the absence of free hydrochloric acid occurs, whilst in cancer it is one of the *earliest signs of the disease*; and this is also true of deficiency in the pepsin. The marked cachexia, emaciation, hæmatemesis, and the presence of a tumour or of pyloric obstruction are all important points in favour of malignancy.

Prognosis.—In the milder cases recovery always occurs when the causes are removed. In the established disease, when atrophy of the glandular tissue has occurred, recovery in the strict sense is not possible, but the patient by judicious dieting may enjoy a considerable degree of comfort, and for long periods may be almost devoid of symptoms. Everything depends upon the extent of motor sufficiency. As long as this is excellent, the food will be driven into the intestine and as complete digestion will occur as is to be met with in any successful case of gastro-enterostomy; but when constant stagnation of the stomach-contents takes place, dilatation and its evil train of symptoms supervene. The prognosis in those cases where the chronic gastritis is secondary to such diseases as tubercle, anæmias, renal and heart affections, is the prognosis of these primary lesions.

Treatment.—Under 'Dyspepsia' the prophylaxis of the transient form of chronic gastritis has been discussed. For the proper management of each case a minute inquiry must be instituted into the causes which are producing the gastritis; thus the nature of the food used, the method of cookery employed, the intervals between meals, rate and efficiency of mastication, the condition of the teeth, &c., are all points of primary importance too frequently overlooked in the routine fashion of writing out a recipe for gastric tonics and handing the patient a printed dietary-sheet. When the chronic gastritis has been of such duration that anatomical changes have been produced in the mucosa, as evidenced by the continual presence of mucus and deficiency in free hydrochloric acid and pepsin, the indication for the administration of digestive agents and lavage is obvious. Lavage is essential in every case where there is evidence of motor weakness in the stomach—*i.e.* where the organ contains food six or seven hours after a full meal. It is necessary in most cases, even when the stomach only contains slimy mucus, as there is little benefit to be derived from drugs, unless this be periodically washed away by patient and thorough lavage. The intern stomach-douche with water at a temperature of about that of the body is very efficacious. The nozzle of the tube should be perforated with numerous fine openings, so that after lavage by the ordinary stomach-tube the entire surface of the stomach may be douched with the finely divided spray. Water at a lower temperature towards the end of the process may be employed with advantage.

This may be combined with Winternitz's method of placing a cold wet compress over the stomach, covering this with a dry cloth upon which are placed coils of rubber-tubing; through these a current of water at 104° F. is made to circulate, or the Scotch douche with alternating temperatures may be employed. The ordinary so-called 'hydropathic pad,' or Priessnitz's compress, consisting of a folded towel wetted with cold or hot water and covered with flannel or oiled silk, may be used.

The drugs usually employed to supplement the feeble powers of the atrophied mucosa are pepsin, papain, and pancreatin, the former being given in combination with hydrochloric acid and the latter two substances with alkalies; papain may, however, be combined with either an acid or alkali.

Pancreatin is useless if the acid and pepsin are not very much reduced or absent, as it is itself destroyed by normal digestion. A teaspoonful of glycerin of pepsin, with 15 mins. of dilute hydrochloric acid and 10 mins. tincture of nux vomica, is often very valuable in simple atrophy. In the early stage of gastritis such a combination would probably aggravate.

Papain 3–5 grs., given with 30 grs. bicarbonate of soda and as much heavy carbonate of magnesia, is never anything but beneficial. As a rule, when the inflammatory action has become permanent, the routine drenching with vegetable bitters does more harm than good, and carminatives are usually injurious also. Creosote may be given advantageously where a mild degree of fermentation is present.

Food should be varied and such as is most easily digested, and in the atrophic stage such as will readily pass into the intestine; hence the fluid or semi-fluid form is to be selected. Thus a good meat-soup is indicated rather than the solid muscular tissue, especially if there be any degree of motor insufficiency. White fish is usually admissible. Carbohydrates and fats may be given in moderate amount, and often cream and butter may be used without hindrance; but the fat of meat, after the prolonged boiling necessary in the process of soup-making, is most objectionable and must be carefully removed. The fat of baked meats is one of the most potent factors in the production of chronic gastritis.

Milk must be used in most cases, and some patients can live only upon a strict milk-diet: it may be peptonised with advantage; but feeding by predigested milk should not be persevered with for long periods. Renneted milk may be substituted from time to time, or the fresh article may be given with alkaline mineral waters. Good buttermilk, when procurable, is a valuable article of food.

Of far more importance than the kind of the food is the way in which it is administered. In cases where the atrophic

changes in the mucosa have led to marked diminution in the free hydrochloric acid and pepsin, the aim of the physician should be to turn the vicarious action of the digestive power of the small intestine to best account. The rational procedure is to administer liquid nourishment *in such small amounts* and *at such short intervals* that it shall pass directly through the pylorus. Thus, if a small wineglassful of milk be swallowed slowly every hour, its digestion will probably be an intestinal affair, whilst a tumblerful every four hours will certainly lead to the tight closure of the pylorus each time, and when the rennet-zymogen is absent fermentation and useless irritation of the diseased organ is produced.

If this method be adopted, all drug-treatment can often be entirely dispensed with, and it is more rational than dosing the useless stomach with pepsin and acid. These may, however, be employed when solids are administered, and it is needless to say that solids should also be given in small amounts and finely comminuted. Lavage must be resorted to daily, as already pointed out, when the food remains for several hours after light meals.

GLANDERS

This is a specific infectious disease of the horse, mule, and ass, caused by the *Bacillus mallei*, and sometimes communicated to man and other animals. The term 'farcy' is used when the disease occurs in the skin or subcutaneous tissues, and glanders when located in the nose or internal organs. It presents itself in both the horse and man in an acute and in a chronic form.

Etiology.—In man the disease is produced by infection from the horse, generally through skin-abrasions; but the normal conjunctiva, and even the unbroken skin, as demonstrated by experiments in animals, may not be a sufficient barrier against the bacilli. Grooms, stablemen, and knackers are usually the victims of the disease, which they may also transmit to nurses or attendants. Veterinary surgeons are sometimes affected. The fine spray, laden with mucous secretion, which is expelled from the nostril of an affected animal upon coughing or snorting may convey the disease for a considerable distance, and vessels used by horses for drinking may likewise spread the affection.

Pathology.—The cause of the disease, as above defined, is always the presence of a bacillus with rounded extremities, which measures in length that of the tubercular organism, but it is much thicker; it does not produce spores, and it grows luxuriantly upon potato, forming primrose-coloured colonies. The bacilli can be procured from the softening

nodules, but cultures from this source are obtained with difficulty. If, however, the peritoneal cavity of a male guinea-pig be injected by the suspected discharge, in a few days a severe orchitis follows, and the bacilli are easily demonstrated in the fluid removed from the tunica vaginalis, and cultures can be readily obtained by this means, which has been used as a test-method by Straus. The bacillus produces a toxin which causes the pyæmic symptoms, and the organism as it grows gives rise to the proliferation of the cell-elements of the infected tissues, resulting in the characteristic nodules, which soon tend to ulcerate and suppurate, causing foul sores.

Morbid Anatomy. — The pathological anatomy of the disease in man corresponds mainly with that seen in spontaneous cases in the horse. The usual site of the proliferating nodules in the horse is the nasal mucous membrane, where they appear as early as the third day after infection. As they break down, a viscid, purulent, or oily discharge appears, which is highly infectious. The growths are also found in all the mucous membranes, and constantly in the lungs and sometimes in all the organs of the body ; the bacilli have been recognised in the blood.

In farcy, which often occurs with glanders, the disease affects the lymphatic vessels under the skin; these form thick cords known as farcy-pipes, upon which nodular growths called farcy-buds develop. Button-farcy is the name given to the nodules which develop in the skin. From all these lesions the bacilli may be obtained.

Symptoms. — In acute glanders in the human subject the incubation is generally three to six days. The symptoms set in with severe fever, joint-pains, and swelling, not unlike severe acute rheumatism. Where the infection has occurred through a skin-wound, this may even heal; but soon erysipelatous redness appears, with involvement of the lymphatics and great swelling, accompanied by crops of vesicles. In a few days, with grave deepening of the constitutional or general pyæmic symptoms, nodules may appear on the nasal mucous membrane; these rapidly break down, causing deep ulcers, which sometimes destroy the bones and give rise to a most fetid discharge. Abscesses form within or between the muscles, especially in the neighbourhood of the affected joints, and large quantities of pus may be evacuated; these abscesses resemble those formed in pyæmia, and in the less acute cases they may be unconnected with the bones or joints, and constitute one of the most characteristic features of the disease. It is, however, not common for the disease in man, even in acute cases, to closely follow the typical glanders of the horse. More frequently it assumes the form of *acute farcy*, in which numerous nodules form in the skin or beneath

it, along the lymphatic routes; these rapidly break down, giving rise to numerous foul ulcers which seldom heal, the patient sinks into a typhoid state after a series of rigors, and death supervenes in less than a fortnight. Before death, even in the pure farcy type of the disease, there is usually evidence of bronchial and of nasal involvement: the conjunctival membrane becomes inflamed and ecchymosed, and great œdema of the eyelids may appear, as in facial erysipelas. In both the glanders and farcy type various cutaneous rashes appear, which are not met with in ordinary pyæmia; thus an eruption like that seen in severe small-pox often invades the face and extremities, and when it appears it is to be regarded as a fatal sign. Bullæ, erythematous patches, and gangrene of the skin may occur. Various pyæmic complications are usually present in the fatal cases, as pneumonia, fetid diarrhœa, empyema, and internal abscesses in several organs, as the brain, liver, and kidney.

Chronic glanders and chronic farcy may run a course of many months; in the former variety the symptoms may resemble those of a chronic rhinitis, but when the septum becomes involved the disease may slowly spread to the pharynx and produce an ulceration of the palate strongly simulating syphilis. The Straus test mentioned upon the previous page, or the injection of mallein, may be the only way by which the disease can be differentiated in such cases.

Chronic farcy is more common in man than chronic glanders, but the lesions vary greatly, though they resemble in the main those seen in the acute form. The subcutaneous or cutaneous nodules may, however, be confined to one locality; the skin over and around them becomes œdematous; as they break down, and suppuration occurs, the indolent ulcers, though deep, may eventually heal, leaving scars. The lymphatic glands usually escape, but hard nodular tumours form on the muscles of the extremities near to the joints; these are usually symmetrically distributed, slowly suppurate, leaving numerous sinuses, discharging thin foul purulent matter. More or less fever and malaise are always present, and the patient may slowly sink from the chronic pyæmia or become the victim of an acute form of glanders or farcy, much after the same manner as acute tuberculosis sometimes supervenes upon a chronic phthisis or bone-disease. In many cases the eruptions mentioned as occurring in the acute forms of the disease appear from time to time; more usually, however, only towards the termination.

Diagnosis.—The occupation of the patient, and the history of his exposure to infection from a horse suffering from glanders or farcy, would seem to be the most important point in the diagnosis; but unfortunately many cases of the

disease in the chronic form in the horse are not recognised or suspected.

These so-called 'latent cases' in the horse are now easily and rapidly diagnosed by mallein. This is a sterilised extract prepared from cultures of the *Bacillus mallei*. The dry preparation is usually employed in doses of $\frac{3}{4}$ gr. dissolved in 80 mins. water, injected subcutaneously. If glanders exists in the animal, the seat of injection swells into a considerable tumour; this increases in size for about thirty hours till a very large swelling forms, which does not disappear for a week. Within eight hours after the dose there is a smart fever produced, analogous to the reaction which occurs in tuberculosis after the injection of Koch's tuberculin; the temperature should rise 2°–4° or 5° F.

It is proposed to use this test in the human subject, but at present the method of Straus seems preferable.

Small-pox, acute rheumatism, acute tuberculosis, typhoid fever, but especially pyæmia, must be differentiated. Goodall lays stress upon the diagnostic importance of suppuration in the muscles.

Prognosis.—This is always grave in acute cases where the percentage of recoveries does not exceed one case in twenty. In the chronic forms of the disease more than 50 per cent. of recoveries are recorded, and the average duration of these is somewhere under six months.

Treatment.—There is no specific remedy known for the disease unless it exists in mallein, but further research alone can settle the question of its value. Many reports seem to show that in mild or chronic cases in the horse recovery has followed repeated injections, and it has been employed with apparent benefit in a few instances of the chronic disease in the human subject. The main indications are to support the vital functions by tonics and abundance of highly nutritious and easily digestible food; each abscess must be opened and disinfected as soon as it appears. All nodules discovered in the chronic form of the disease should be promptly excised and antiseptics freely applied. Where infection has occurred through a cutaneous wound, the site of this should be cut out without delay.

Prophylactic measures are obvious, and need not be detailed.

GLANDULAR FEVER

This is an acute, infectious, febrile affection of childhood, often occurring in epidemic form, and characterised by a mild inflammation of the lymphatic glands of the neck and other regions.

The disease was first described by Pfeiffer, and many epidemics have been recorded since his published report in 1889. Adults are very rarely affected. The period of incubation is usually about one week, and is followed by a sudden invasion of mild fever, setting in with headache and pain and some stiffness on moving the head. The tissues beneath the skin of the anterior triangle of the neck are tender on firm pressure. Usually there is some nausea or vomiting present, and an early symptom, according to Dawson Williams, is abdominal pain and tenderness, usually associated with marked constipation.

The throat may reveal trifling signs of pharyngeal catarrh, which are not of such a character as to explain the pain which is often produced by the effort of swallowing. Upon the second evening or the third day the glands below the sterno-mastoid muscle are found to be enlarged and tender, usually first on the left side. Other glands become involved, so that all the cervical group on both sides may be noticed as swollen and tender. The axillary and inguinal are often involved, and the mesenteric group can be felt through the abdominal walls in a considerable percentage of cases. The fever reaches its highest point about the third evening, when the glands are first noticed as tender and swollen; usually it marks from 102°–104°, but soon subsides as the tumours diminish in size and tenderness. Some enlargement of the liver and spleen may be noticed, and a tickling cough is sometimes present.

By the end of a week or ten days all fever has usually disappeared and the glands are steadily diminishing in size, but they often may be felt to be still somewhat enlarged for three or four weeks. They never suppurate; when such has been noticed the condition was probably due to some other disease. In one outbreak of the affection in a large industrial school the fever was so mild as to be overlooked; and when coming under the writer's observation for the first time about fifty cases were found in which there was no other sign or symptom but the glandular enlargement, which entirely subsided in the great majority of these within fourteen days, but in about 10 per cent. of the cases the glands were still to be detected enlarged, but not tender, after six weeks. In one case very great enlargement of the glands in front and behind the sterno-mastoid remained for four months, when a tubercular adenitis was suspected as a coincidence; but they suddenly began to diminish, and within a week could scarcely be felt.

Pathology.—Dawson Williams is inclined to regard the pathogeny of the disease as an infection from the intestinal surface. It is probably a specific fever, but the micro-organism causing it has yet to be demonstrated. The early involvement of the mesenteric glands is suggestive of the intestinal route for the infective agents. The prognosis is invariably

good, all cases recovering; but it is just possible that the affection may have its own sequelæ which appear at a later date, and are not yet recognised.

Treatment.—Nothing is required in the majority of cases but a simple diaphoretic with an occasional mild saline purge. The less interference with the glandular enlargement the better, and friction should not be used.

GLÉNARD'S DISEASE.

(See ENTEROPTOSIS, p. 383.)

GLYCOSURIA.

(See under DIABETES, p. 273.)

GOÎTRE

This term, or its synonym Bronchocele, is applied to enlargement or hypertrophy of the thyroid gland.

The subject is one belonging to the domain of surgery, but the intimate relationships of the thyroid gland to cretinism, myxœdema, and Graves' disease, which are discussed under the headings of these affections, justify a brief reference to the enlargements of the gland in a work devoted to medicine. This will be found in the article on 'Thyroid Gland Disease.'

GOÎTRE, EXOPHTHALMIC

Also known as Graves', Parry's, and Basedow's Disease. This is an affection characterised by thyroid enlargement, exophthalmos or protrusion of the eyeballs, tachycardia, and muscular tremors.

Etiology.—Women are affected much more frequently than men, and Gowers states that the ratio is about twenty to one. Murray gives it as eleven to one; other observers give very varying figures. The disease being rare in men, a much larger proportion of male cases is placed on record.

The most common period of life for the symptoms to first show themselves is between the ages of twenty and twenty-five years, but no age is exempt. Heredity is not a marked feature in the etiology of the disease, but it is not uncommon to find that two or more members of the same family have suffered from it; generally a neurotic history is obtainable, and in this way it is found indirectly associated with epilepsy, insanity, hysteria, and occasionally with diabetes.

Severe mental emotion, as sudden fright, prolonged excitement, grief, or worry, is a frequent exciting cause, and occa-

sionally it has followed violent muscular exertion and recovery after acute illnesses, especially influenza and acute rheumatism. According to F. T. Roberts, it is almost always associated with the anæmic and chlorotic states and menstrual disorders when occurring in young females.

Pathology and Morbid Anatomy.—The pathological anatomy of the disease shows a general and usually uniform enlargement of the entire gland, which is firm and elastic. On section there is found hypertrophy of the secreting structure, and the changes may be taken as representing those which might be expected in a gland in a state of hyperactivity. The thymus is usually found to be enlarged and extending downwards, covering the upper portion of the pericardium. In some cases the enlargement is confined to one lobe, and adenomatous or cystic growths may be found associated with the general hypertrophy.

The heart is usually dilated, and valve-trouble may be present in some cases; and occasionally the sympathetic ganglia have been found diseased.

The pathology of the disease is still awaiting solution; but it would seem almost certain that, whatever unknown factor may be present, the majority of the symptoms are produced by an over-active state of the thyroid, whereby its secretion is greatly increased. This state, to which the name 'hyperthyrea' has been given, is considered by Möbius and others to be the opposite to the myxœdematous condition; and that this is correct is almost proven by the fact that thyroid feeding almost always, if not invariably, aggravates the majority of the symptoms. But though large doses of thyroid gland administered to healthy subjects will produce a condition resembling Graves' disease, they will not cause the eye-symptoms. It seems, therefore, that some other factor is at work, and two probable causative agents have been demonstrated recently by experimental methods.

Thus Tedeschi and Filehne have succeeded in producing the disease in animals by causing lesions in the restiform bodies, and the symptoms were removable by excising the thyroid.

Booth urges that the connecting fibres (sympathetic and vagus) between the central nervous system and the gland must be also taken into account. This latter view is proven to be correct by the large number of cures of exophthalmic goître which have been effected by removal of the cervical sympathetic. It would therefore appear that the best explanation of the pathology of the disease is that held by Booth, in which the three factors are recognised—viz. the central nervous system, the connecting fibres, and the gland itself—a lesion in any one of these parts probably producing an alteration in the others which, in conjunction with the exciting

cause, would produce the disease. Salmon maintains that the disease originates in some disturbance of the hypophysis cerebri, whose internal secretion is diminished, and this leads, in his opinion, to functional over-activity in the thyroid. That the affection is a neurosis, a blood-condition or auto-intoxication, or a disease of the parathyroid, has been held by various authorities.

Symptoms.—The onset is generally gradual, though in rare cases the disease does set in suddenly with acute symptoms. The first symptom to appear, according to most authorities, is the rapid action of the heart; whilst some state that the enlargement of the thyroid appears first in the majority of cases. The question of the priority of the symptoms depends upon the patient's temperament. Some attach little importance to cardiac frequency, but speedily notice any change in the contour of the neck; many patients only seek advice for one symptom, whilst two or three may be present without their knowledge. Upon the whole it is correct to say that in the majority of cases the palpitation first appears, increases slowly, and is followed after a variable short interval by the almost simultaneous advent of exophthalmos and goître.

Heart.—The beats range between 100 and 140 per minute, and in the severe cases they may reach 180; the writer has counted 200 once. The impulse is strong, visible, and always palpable over a larger area than the normal; the action is regular. In the later stages irregularity is not uncommon. The apex is displaced outwards to a considerable extent, and actual dilatation is then present.

Systolic murmurs are often heard with the loud cardiac sounds, either at the apex or pulmonary area and often in both situations; occasionally the systolic murmur is audible over the entire cardiac area and beyond it, and synchronises with a thrill in some cases.

The tachycardia is markedly increased by excitement and exercise, often to an extent which causes dyspnœa. Blake maintains that often the heart acts most rapidly when the patient is in the horizontal posture.

Visible pulsation or throbbing is almost always obvious in the neck-vessels, which appear much dilated, and it may extend to arteries at a distance, and loud murmurs may be audible in them.

Thyroid.—Enlargement is almost always present, but in a considerable number of cases (12 per cent.) it is stated by Murray that some goitrous enlargement of old standing had preceded the development of the symptoms. The increase in the size of the gland is uniform as a rule, but it is not unusual for the enlargement to commence in one lobe, The thyroid vessels throb, and often the entire gland pulsates; murmurs

are frequently audible over its area ; sometimes a distinct thrill can be felt, and the superficial veins may be distended. As a rule the amount of enlargement is not very great, rarely is it seen to be as marked as is common in many simple goîtres. The size of the gland seems to vary at times, and usually increases on excitement.

Exophthalmos.—This naturally is the most striking point in the clinical picture, and hence in a considerable number of cases it is stated to have been the first symptom noticed ; but it is rare to find it marked before the advent of the two previously mentioned signs. According to Murray's figures, it was absent in rather more than one case out of every four in 114 cases specially examined for this sign. It is almost invariably bilateral, but not rarely it is better marked in one eye than the other. Often it is so extreme that when the heart is acting violently the eyeballs appear to be starting from their sockets, and even the insertions of the recti muscles become visible. The lids are unable to cover the globe, so that the white sclerotic is evident beyond the corneal margins, and the patient seldom winks. Actual dislocation of the eyeball has occurred and sloughing of the cornea and other structures has been reported. Swelling of the lids and conjunctival chemosis are occasionally present. Vision is normal, and the pupils show no change ; the ophthalmoscopic examination usually reveals only marked pulsation in the retinal arteries and occasionally in the veins.

Five other signs have been described as often present : (1) Von Graefe's. This is elicited by asking the patient to watch any small object held in front of the eyes. As the object is moved downwards it will be noticed that the upper lid lags behind and does not follow the movement of the eyeball, being delayed or hindered in its descent by a variable degree of retraction which causes the sclerotic to remain visible above the upper margin of the cornea. When well marked this sign is unmistakable ; but it is in the writer's experience often absent, or only present in such trifling degree as to be doubtful.

(2) Stellwag's sign is often confused with the above, and described as a retraction of the upper lid. It really consists in an increase of the palpebral fissure, and does not depend upon the extent of the exophthalmos, but is the result of the retraction of the lid, and, as pointed out by Gowers, Von Graefe's description included retraction.

(3) Möbius's sign is of less importance. It is recognised as a loss of power in the internal recti muscles in converging for near objects.

(4) Joffroy's symptom is sometimes present. It is elicited by causing the patient to flex his head upon the chest, and when in this position he attempts to look upward there is no wrinkling of the forehead.

(5) Jellinck maintains that there is an early eye-sign frequently overlooked, as it disappears or becomes less marked at a late stage ; this consists in a diffused brownish pigmented discoloration, most marked in the upper eyelid, and which does not extend into the eyebrow above or pass below the inferior margin of the orbit.

Tremor.—This is the fourth characteristic feature in the clinical picture ; it varies much in degree in different cases, and is absent in but a small proportion of the examples of the disease when carefully sought for. Thus it was present in 111 out of 120 of Murray's cases. It is a fine regular tremor which affects the entire upper extremity, and may be brought into evidence by directing the patient to stretch out his extended hands in front of the body. It is also often present in the legs, and is usually bilateral ; but it may be present at one time and absent at another, and with it a coarse jerky movement of larger amplitude not very unlike that seen in chorea may be sometimes observable.

With the above described cardinal symptoms are usually found many others apparently entitled to equal rank. *Nervous disturbances*, as irritability and restlessness and continuous fidgetiness, but especially a chronic state of pent-up excitement or expectancy, are common ; and occasionally insanity in the form of mania or melancholia develops. *Weakness* of the leg-muscles of almost paraplegic character is a not unfrequent symptom, and causes the patients often to fall or sink down ; and the majority of them complain of being very easily tired.

Emaciation may be as extreme as that observed in diabetes or cancer, though there is no elevation of temperature or loss of appetite ; mild cases, however, do not usually show much failure of nutrition, and in acute examples of the disease one of the first signs of improvement may be a steady gain in weight. *Anæmia* is occasionally extreme, and menstrual irregularities are frequent.

Cutaneous changes are common ; one of the most remarkable of the phenomena of the disease is the great diminution in the electrical resistance when the current is applied to the skin. This is explained by the constant increase in moisture caused by over-activity of the sweat-glands. Pigmentary changes are common on the exposed portions of the forearms, hands, and face ; the bronzing may resemble that found in Addison's disease, and is sometimes associated with leucodermia, and it may exist in patches scattered over the trunk and limbs. The hair gets thin, and is shed completely in some cases, but returns on recovery. There are often great flushings, urticaria, and pruritus.

Digestive troubles are noticeable in many cases ; thus the appetite may be abnormally great or perverted, or lost in the

late stages. Vomiting and diarrhœa are common, and may prove grave complications.

Respiration may be much quickened, and dyspnœa is often seen, especially on excitement or exercise, though the respiratory movements are diminished in depth.

Urine.—There may be glycosuria and intermittent albuminuria; rarely, however, do these pass into diabetes or renal disease.

The course and duration of the disease is extremely variable; some cases begin acutely and end in recovery or in death in six or eight weeks, whilst others drag out an existence for very many years. There is a distinct relapsing type in which recovery is *almost* complete in the long intervals between the attacks. Though recovery is the rule, the symptoms often last for years, and there are vestiges of neurotic instability, irritability of the heart, and some enlargement of the thyroid, with a slight staring expression in the eyes, left permanently or for very lengthened periods.

The writer has seen one case followed by myxœdema after an apparently complete recovery, and symptoms very like the typical disease set in after large doses of thyroid had been taken for three months; these subsided on stopping the drug, and small doses afterwards were well borne.

Diagnosis.—When the four cardinal symptoms are present there cannot be a difficulty. But *incomplete* forms are not uncommon, in which, for example, there may be absence of exophthalmos and tremor; and the diagnosis must then be verified by a careful search for the general symptoms already detailed, especially for evidence of mental or nervous disturbance. Though the exophthalmos is the most obvious of the symptoms, from a diagnostic point of view in the incomplete forms the cardiac frequency is of more importance. It may well be doubted if the disease ever exists without increased frequency of the heart-beats, though it certainly may be present without thyroid enlargement to any appreciable extent.

There may be a difficulty in the diagnosis of those cases already alluded to where the disease has supervened upon an old goître, but the fact that many such have been recorded should remove all hesitation in the presence of tachycardia. A patient at present under the writer's observation, and seen recently by Professor Allbutt, developed mild exophthalmic goître after suffering for a few years from a typical simple goître. A considerable percentage of such cases is probably due to the administration of thyroid extract, prescribed for the original goitrous enlargement.

Hysteria should not be mistaken for exophthalmic goître, and the mistake can only occur when hysteria is associated with the disease, which is often.

Prognosis.—It is most difficult to forecast the issue : cases which set in acutely soon after severe fright may be quite well in a few weeks. As a rule, however, the symptoms setting in in the acute form should lead to a serious prognosis, unless in children. Taking all types of the disease into consideration, it is not likely that the mortality exceeds 15 per cent., but the death-rate in reported cases is much higher, since severe cases are recorded in larger proportion ; many patients, however, are lost sight of. It must be also remembered in the prognosis that death may unexpectedly occur from syncope, severe diarrhœa, or vomiting. Great emaciation and a pulse-rate of over 160 are grave symptoms ; so also is mania. Intercurrent diseases like acute pneumonia and acute bronchitis are often fatal, since cardiac dilatation is nearly always present.

Treatment.—Rest in bed is essential in all severe cases with very rapid pulse. Drugs are most disappointing, though nearly every known drug has been vaunted at some time or other as curative. The best results are obtained from full doses of strophanthus or digitalis. Belladonna has been always useless in the writer's hands, and bromides also have proved valueless.

A weak continuous current applied over the cervical sympathetic is unquestionably beneficial, especially if combined with the passing of a weak current through the brain, with one electrode over a pad of warm wet lint placed on the closed eyelids and the other on the occiput.

Ice or Leiter's tubes over the thyroid for hours at a time may reduce the severity of the symptoms temporarily.

Thymus, adrenalin, and testicular extract have their advocates, but thyroid feeding should never be employed.

Working upon the theory that the thyroid function is to neutralise toxic metabolic products, many observers have reported most encouraging results from administering the serum of thyroidectomised sheep, and Lanz employs feeding by the milk of goats whose thyroids have been previously removed by operation. Burgharts and Blumenthal isolated the active ingredient in the milk as an alcoholic extract, under the name of *rodagen*, which is claimed to possess curative powers.

Operative treatment has been frequently carried out of late years, and with marked success. The excision of the enlarged gland is successful in more than half the cases, but a portion should be left behind. The mortality is high.

Removal of the sympathetic nerve with the three ganglia on both sides is decidedly the most certain remedy yet introduced, and the mortality is lower than that of thyroidectomy. Jonnesco has performed this operation fourteen times without a death. Only one side should be operated upon at a time, and local anæsthesia is much safer than chloroform or ether.

GOUT

This is a constitutional affection whose chief manifestation is an acute form of arthritis, which is characterised by the deposition of crystalline sodium biurate in the interior of the affected joints, in the fibrous tissues in the immediate neighbourhood, and in the fibrous tissue in other regions of the body. Its old name, *Podagra*, derived its origin from the frequency with which the metatarso-phalangeal joint of the great toe was affected.

Etiology.—*Age.*—The classic period at which the *first attack* of gout appears is about the age of thirty-five years. Owing to the chronic nature of the disease, gout is too often associated in the mind of the physician with a much later age-incidence, since the great bulk of the patients complaining of chronic gouty symptoms are long past middle-life. A first attack may occur in early adult age, but this is very rare; whilst it is not uncommon to meet with first attacks at the ages of sixty, seventy, or even later.

Sex.—The disease occurs with much greater frequency in males, being very rare in women.

Heredity.—This is strongly marked in the disease, over 60 per cent. of the cases giving a family history of direct inheritance. Where this predisposing factor was well marked in the patient's history, first attacks have been recorded in boys under twelve. Roberts states that though the women in gouty families escape, the transmission of the disease to offspring is more certain through the female line.

Hutchinson points out that the children born of a gouty parent in his early life frequently escape, whilst those born after the parent has advanced in life frequently suffer severely.

Habits.—*Alcohol* is a potent factor in gout—more strongly operative perhaps than heredity; and it is certain that the form in which the alcohol is taken has much more influence than the alcoholic strength of the beverage. Fermented liquors, like Port, Marsala, Champagne, Burgundy, Madeira, ale and stout, even cider, operate strongly as causative agents, whilst distilled spirits—whisky, gin, and brandy—have little effect; but rum undoubtedly may induce the condition. Hence the disease prevails in certain occupations, as amongst brewery operatives, whilst in those working in distilleries it is rare. It is common in the wine districts and beer countries, and unusual in places like Ireland and Scotland, where spirits are the fashionable beverage. F. T. Roberts states that indulgence in a mixture of alcoholic drinks is more likely to lead to gout than when any particular wine is adhered to.

Food.—The nature and amount of food constantly used is a powerful factor. Gout is a disease produced by high living. Rich, highly nitrogenised animal food, where taken in large amounts in conjunction with fermented liquors, by those with hereditary taint, seldom fails to produce the disease. Even great feeders who take but a small amount of rich animal food generally escape gout.

Exercise is possibly as strong a factor as temperance in preventing the onset of the disease in those predisposed. Indolent habits, when combined with gross feeding, are a powerful exciting cause; hence the disease is certainly most frequently met with in those members of the wealthy and leisured class who do not freely indulge in active open-air sport involving abundant muscular exercise. It is not, however, rare amongst active draymen and cellarmen, who take large quantities of beer and porter, and who have to work hard for their living.

Lead-poisoning operates actively as a cause, especially when combined with the previously mentioned etiological factors, but it probably is of much less moment when it is a sole cause. The connection of gout with kidney-disease is usually through lead-impregnation, which causes the so-called 'gouty' or granular form of Bright's disease. Dyspepsia is very frequently present in gout, and is by some regarded as an exciting cause.

Symptoms.—A typical attack of gout is usually suddenly ushered in with clearly defined symptoms of an acute inflammation attacking the metatarso-phalangeal joint of the great toe. The seizure is most common in the early hours of the morning, and may follow an unusually heavy dinner of rich or highly nitrogenised animal food, accompanied by copious libations of wine; or it may be some trifling injury or exposure to cold, worry, or fatigue which lights up the latent vice of metabolism which had been slowly maturing. There may, however, have been *premonitory* symptoms for a few days before, in the form of dyspepsia, palpitation, restlessness, peevishness, or depression.

The pain in the affected joint is severe from the first moment in which it startles the patient from his sleep, and it rapidly becomes agonising. In a few hours the joint becomes tensely swollen, red, and exquisitely tender, and the slightest attempt to move it increases the pain. The suffering has been often described as like that which might be produced if the joint were placed in a vice and the screw turned on tightly. The constitutional disturbance is severe, though the temperature may not exceed 101° or 102.° The urine is scanty and high-coloured, the tongue dry, and the mouth parched. As the day advances there is usually some relief experienced, and the patient may get a few hours' broken

sleep; but as evening approaches the pain returns and may become more excruciating, and may last all through the following night with its accompanying fever and restlessness.

The swelling around the joint increases, and the veins under the shining tense skin become evident. As the tenderness slowly diminishes, the tissues may be observed to pit on gentle pressure.

By the end of about a week the swelling and other symptoms have disappeared, and in first attacks the recovery usually is apparently complete in about ten days. Desquamation of the skin about the affected joint so constantly follows that it is an important diagnostic sign. Suppuration may be said never to occur. Other joints may, however, be affected in 'the fit,' as it is called; thus, the gouty inflammation may extend to the neighbouring joints, and sometimes the opposite great toe becomes involved, prolonging the duration of the attack by another week or ten days of suffering.

Recurrences are the general rule; but after the first attack the patient's health is usually better than it had been for a long time previously, and he may not experience another seizure for several years. The length of the intervals between the attacks depends upon the patient's habits. When he turns over a new leaf, regulates his diet, abstains from all fermented liquors, and takes abundant open-air exercise with unfailing regularity, he may never experience another attack unless the taint of heredity be strong. Usually, however, after a few years the attack returns in the same joint, or in that of the opposite toe; and as the patient advances in life the tendency is for the seizures to become periodical—at first once or twice a year, and finally in inveterate cases more frequently involving new joints and returning to those first affected. In this way all the small joints of the feet and hands, the ankles, wrists, and knees, become involved; the hip, shoulder, vertebral and jaw articulations usually escape, unless they have been the seat of injury previously. At this stage the patient has scarcely recovered from one attack till signs of another seizure appear; the disease loses its periodic characters, and the condition recognised as *chronic gout* may be said to have become established, in which visible changes in, and obvious deformities of, the affected articulations are constantly observed.

The joints in chronic gout become the seat of chalky deposits or tophi, consisting of biurate of sodium. This is first deposited during the gouty paroxysm in the superficial layers of the articular cartilages in crystalline needles; subsequently the fibrous structures around the joint, the tendons, ligaments, bursæ, and subcutaneous tissues become infiltrated with the deposit, so that masses of various dimensions form around the articulation; and as the red, thinned, and shining skin

ulcerates over them, the so-called gout-stones become exposed, and may be cast off. More frequently, from the cutaneous ulcers caused by their presence, a creamy white liquid exudes, which consists mainly of the needle-like crystals of the sodium salt mixed with serum and pus.

Tophi may also be found in the fibrous tissues away from joints, as in the palms of the hands and soles of the feet, or in the sclerotic. In one easily accessible region they form with such frequency that their presence should be always sought for, and when found it constitutes a valuable diagnostic sign of the disease. This is under the skin covering the cartilage of the helix of the ear, where they may be recognised as small, hard, bead-like projections of a pearly-white colour. If observed in their early stages, as described by Garrod, they are of a soft creamy consistence.

The deformity of the hand caused by the gouty deposits about the joints resembles somewhat that observed in chronic rheumatoid arthritis. Thus the fingers may be deflected towards the ulnar side, and the joints more or less anchylosed in both affections; but the presence of the white chalky deposit under the skin, especially if this has ulcerated, is pathognomonic of gout.

The condition of the blood and urine in gout will be discussed under the Pathology of the affection. When there is granular kidney, albuminuria, cardiac hypertrophy, rigidity of the vessels, increased pulse-tension, and the other symptoms of arterio-sclerosis will be present.

IRREGULAR GOUT

This form of the disease is rare in the subjects who suffer from regular periodic fits until these have passed into the chronic stage, when in the brief and irregular intervals various symptoms are complained of, which are grouped together under the name of irregular gout or the *uric-acid diathesis*, *lithæmia*, or *suppressed* gout. These symptoms are quite common in the members of gouty families who have never suffered from arthritic seizures or shown urate-of-sodium deposits, and the term is a favourite one with some physicians and most patients in innumerable conditions totally independent of gout, where a diagnosis is wanting. Many patients feel flattered and proud of any ailment which they can call gouty.

Skin-eruptions, especially a form of dry scaly eczema which affects the ears and face in itchy patches, is very common in gouty patients, and very resistant to all treatment; it is often mistaken for psoriasis.

Nervous disturbances, as neuralgia, migraine, and neuritis affecting the sciatic nerve or the peripheries of the plantar or palmar nerves, are often met with, and the peripheral

irritation has been believed to be the forerunner or cause of epilepsy in some cases. A restless, irritable, or depressed mental condition is common, which sometimes causes a severe form of insomnia aggravated by painful cramps in the calf-muscles.

Heart-symptoms are often complained of, independent of those proper to the accompanying renal lesion, and present when this is absent. Irritability of the nervous mechanism of the heart best describes the condition in which syncope, anginal symptoms, palpitation, and tachycardia may alternate. Thrombosis is not uncommon in both veins and arteries, and may be the cause of a fatal apoplexy.

Bronchial irritability is seen in a dry form of chronic bronchitis, which often is associated with symptoms of asthma, and not infrequently an asthmatic seizure precedes an acute attack of gout.

Eye-troubles, as sclerotitis, iritis, glaucoma, and retinal hæmorrhages, are common.

Urinary symptoms, independent of organic renal disease, are often met with. The output of uric acid is usually, but not always, reduced in chronic gout; occasionally the acid is eliminated in unusually large quantities, when the mechanical irritation caused by the acicular crystals produces severe irritability of the urinary tract. In one case under the writer's care there was a sudden, severe, and *purulent urethritis*, with very copious discharge of pure pus, which exactly resembled a very acute attack of gonorrhœa; the diagnosis of gout was corroborated by Burney Yeo, who saw the patient during a subsequent sudden attack.

Glycosuria, albuminuria, oxaluria, and more rarely urinary calculi, are met with; but often the urine may be quite normal, both in quantity and quality, and the excretion of uric acid may exhibit no deviation from that of typical health for very long periods.

When the granular cirrhotic stage of renal disease becomes established, all the signs and symptoms described under Bright's disease are met with.

Gastric and *intestinal disturbances* are so constantly met with that a form of gouty dyspepsia is recognised by most clinicians. Murchison, who regarded gout as the result of liver-torpidity, described the hepatic discomfort, flatulent distension, acidity, constipation, diarrhœa, &c., under the titles of 'lithuria' and 'lithæmia.'

RETROCEDENT GOUT

This is the name given to the phenomenon which occurs when the gouty inflammation suddenly leaves an acutely affected joint and attacks some internal organ, as the heart. Such a rapid metastasis of the articular mischief to the heart

may cause violent palpitation, associated with grave irregularity, and a fatal syncope. When the gouty mischief flies from a joint to the brain, meningeal symptoms may rapidly develop, ending in delirium and coma. The stomach may be the region selected, and then violent pain and gastritis supervene, which may prove fatal. The symptoms are not so grave when the arthritis flies from one joint to another joint.

Morbid Anatomy.—The characteristic deposits around the joints, as studied at the bedside, correspond to those seen after death. The morbid conditions in the interior of a recently opened joint are visible to the naked eye: the white chalky deposit of sodium biurate is seen incrusting the articular cartilages, capsules, ligaments, and synovial pouches, and in advanced cases the deposit, as already described, is found invading all the tissues outside the joint, including the skin, and tophi as large as a small orange have been found.

A thin stratum on the surface of the cartilage is always free, a section showing that the deposit is immediately below this, extending downwards towards the bone, which, however, with the lower layers of the cartilage, is always free from the crystalline deposit. The cartilaginous matrix is profoundly altered or necrosed in its upper layers; but it appears certain that the changes are secondary to, and caused by, the mechanical injury due to the deposited crystals, and not, as taught by Ebstein, to a primary necrotic change in which crystalline needles are afterwards deposited. It is noteworthy that the densest patches of crystalline deposit are always found in the zone of the cartilage furthest removed from the blood-supply, and hence Roberts infers that their origin is from the synovial fluid bathing the interior of the articulation. Though the presence of these deposits may be taken as proof that a gouty inflammation had previously attacked the joint, as insisted upon by Garrod, they are sometimes noticed to a lesser degree in joints which have never been the seat of pain or obvious inflammatory action. Roberts explains the occasional absence of the uratic deposits in joints which were known to be gouty by the supposition that the crystals had been absorbed in the interval following the attacks.

Sometimes the deposits have been found by Norman Moore in all the joints of the lower extremities, when entirely absent from those of the upper limbs. The renal changes are much less constant than the articular, but it is not uncommon to find the *post-mortem* evidences of the so-called 'gouty kidney' of chronic Bright's disease. Probably these are always found in cases where lead-poisoning was a factor. In not a few instances the arthritic manifestations have been known to appear only after the renal condition had previously existed for a lengthened period, in which case the gout may be considered

to have been long latent; and this view is rendered probable by another fact noticed—viz. that the autopsy in cases of granular kidney has revealed a gouty deposit in several joints when arthritic manifestations were absent from the history of the affection. The deposit of biurate-of-sodium crystals may be often found extending in streaks between the tubules in the pyramidal region and dotted irregularly in small areas throughout the cortex.

The cardiac and vascular changes are those seen in arterio-sclerosis when renal disease is a portion of the pathological anatomy of gout.

Pathology.—The pathogenesis of gout is a mighty maze which, in the chaotic state of our imperfect knowledge, often seems to be without a plan. Little progress can be expected towards a solution of the difficulty till the large mass of crude theories and imperfectly digested facts are ignored and a fresh start is made by experimental methods and patient clinical investigation.

That gout is manifested by or associated with a deposit of biurate of sodium in the affected joints is obvious. That this deposit is the result of a perverted metabolism, the cause of which may be either inherited or acquired, seems certain.

The important question simply put by Woods Hutchinson—Is the presence of uric acid or urates in the blood and tissues the *cause* of gout, or merely a *symptom* of the disease ?—requires a definite answer which is not yet forthcoming. The researches of Roberts, Garrod, and a host of others on the chemistry and source of the uric-acid compounds have not solved the difficulty. A solution may possibly come from an unexpected quarter. The action of colchicum in gout seems a remarkable one, about which, however, practically nothing is understood or known. The drug, though called an alterative, and sometimes a specific, still remains a typical example of a purely empiric remedy. When the pharmacology of colchicum becomes fully worked out, we shall not be far from a knowledge of the pathology of gout.

The sources of uric acid at present are believed to be, in the normal individual, two: one portion is derived from the nucleins and purin-bases of the food (Chittenden), while a quantity is derived from the breaking-up of the nucleins existing in the leucocytes and other cells (Levison).

Woods Hutchinson maintains that in health the exogenous source of uric acid—viz. that portion derived from the purin-bases of the food—accounts for a much larger amount of the acid than is derived from the endogenous supply furnished by the destructive metabolism of the cells of the tissues, and in gout he believes that it is the latter only which is increased; but, as will be presently mentioned, he considers the presence of the increased acid as but a symptom of a general toxæmic

process. The purin-derivatives can be converted into uric acid by ferments which have been extracted from the spleen and liver.

Xanthin and hypoxanthin constitute the oxy-purins ; they are found in muscle, and meat-extract is very rich in these bodies. After their administration the excretion of uric acid is always augmented. The amido-purins are derived from pancreas and from thymus, and the ingestion of these foodstuffs very considerably increases the excretion of uric acid through the breaking-up of their nuclein. The methyl-purins, represented by caffeine and theobromine, are of little importance, since the uric-acid excretion is not increased by their administration.

Tidy has shown that after a purin-free diet, consisting of eggs, fats, carbo-hydrates, white bread, and potatoes, the excretion of purins goes on continuously—a fact which demonstrates their endogenous source from the nuclein of the leucocytes and other cells in the tissues.

It is unnecessary to discuss whether the seat of the manufacture of uric acid is to be located in the kidney, as held by Luff and Garrod ; or in the liver, or by a harmonious working of both these organs, as believed by Latham ; or whether the part played by the kidney in the *rôle* of uric acid is simply to destroy it, as held by Crofton.

That uric acid exists in the blood in gout, and not in the blood of the healthy individual in any appreciable quantity, is an important consideration. This discovery was made about sixty years ago by Garrod, and since then, unfortunately, its natural result has been to associate ' gout ' and ' uric acid in the blood ' as synonymous terms—which is unquestionably an error, and one which has retarded rather than advanced the progress of research towards a final solution. Garrod accounted for the presence of the acid in the blood by a diminished elimination of it by the kidney.

Garrod's method of detecting uric acid in gouty blood is a simple one. Two drachms of the blood-serum, mixed with twelve drops of strong acetic acid, are placed in a watch-glass with a few fine threads detached from a linen fabric, and the serum is allowed slowly to evaporate almost to dryness by placing the glass for a day or two in a warm place. Under the microscope minute rhombic crystals of uric acid are observed on the threads. The serosity from a blister applied at a distance from the gouty joint always gives the same reaction.

The next point in the consideration of the gout-problem is the deposition of the acid in the tissues. Garrod maintained that this was caused by the diminished alkalinity of the blood-serum, which became so weak as a solvent that it permitted the acid to crystallise out. This hypothesis is now

generally considered to be untenable, though still its correctness is affirmed by Haig.

Roberts explains the gouty deposition from the blood in a manner which has met with wide acceptance: the uric acid never exists in the blood as a free acid, but as a urate. He points out that there are three classes of urates—the *neutral urates*, which never occur in the body; the *quadri-urates*, which is the form in which uric acid is always found in normal urine; and lastly, the *bi-urates*. This is the form in which uric acid is always found in the gouty concretions.

The uric acid in the blood in normal states, according to Roberts, exists as a quadri-urate in small amounts, which are eliminated by the kidney, preventing uratæmia, or accumulation of the acid in the system; but in gout the elimination is interrupted by deficient action of the kidney, or excessive formation of urates. The retained quadri-urate is changed in the blood by the normally present sodium bicarbonate into the biurate, which, in the gelatinous or hydrated form, continues to circulate till it becomes changed into the almost insoluble amorphous or crystalline form, which is thus deposited in the tissues. Biurate of sodium at blood-heat is soluble in about 1,000 parts of pure water, but the addition of ·7 parts of sodium salts renders it practically insoluble. This proportion of sodium salts maintains in the normal serum, hence the biurate is precipitated. In this way Roberts explains the gouty arthritic paroxysm, which he believes to be caused by the sudden precipitation of acicular crystals in the cartilagenous and fibrous structures of the joint. A study of the relative richness of the various fluids and tissues of the body in sodium salts supports his theory; thus synovial fluid contains ·8 and cartilage ·9 per cent. of sodium salts, whilst muscle, in which gouty concretions are never seen, contains but ·08 per cent. Blood-serum and lymph contain ·7 per cent.

The uric-acid theory—viz. that gout is but the direct result of an accumulation of urates in the blood whose precipitation causes all the phenomena of the disease—at first sight so plausible, is inconsistent with other facts. Thus, excess of uric acid in the blood is frequently observed in many other diseased conditions in which gouty symptoms are never seen, as in anæmia, pneumonia, and especially in leukæmia, where the excess is abundant and constant, but always harmless, no deposition of biurate or gouty paroxysm occurring. Another very important fact has been frequently demonstrated which strongly militates against the uric-acid theory when applied in its usually accepted simple form. Ibrahim, Soetbeer, and others have injected uric acid in large amounts subcutaneously, and found that it was not only all excreted by the urine, but that for days afterwards it caused an increased elimination of the acid, which must have been the

result of increased formation within the body. The acid has been administered by the mouth in large doses, alone or mixed with the food, and no toxic effects have been noticed, so that it is now regarded by many to be absolutely non-irritating, except, perhaps, when appearing in the crystalline state in the urine it may cause temporary mechanical irritation of the bladder or urethra. Moreover, abundant evidence is forthcoming that there is no necessary connection between the production of urea and uric acid. (It is established that there is a close relation always between the excretion of uric acid and phosphoric acid in gout—Futcher.) Thus, though it may be accepted almost without reservation that an attack of gout cannot take place unless there is an accumulation of urates from retention in the blood, it is obvious from the previously mentioned facts that there must be some other factor at work which determines the deposition of the biurate in the tissues. Hence a host of new theories have been invented for the explanation of the pathology of gout. The trend of opinion seems to be in the direction of regarding the presence of uric acid in the blood and its deposition in the tissues as but a symptom of some grave error in metabolism, probably caused by a toxin. In the search for the origin of this problematical poison the intestinal canal has been honoured by many workers in the gout-field. Auto-intoxication, either from a ptomaine produced by perverted digestion or by bacterial activity (Chalmers Watson and Gore) or some putrefactive changes in the intestinal contents (Thorne), is regarded as a good working basis upon which to explain the protean manifestations of the disease. Woods Hutchinson, Levison, and others explain the presence of uric acid in the blood as the mere measure and result of the destructive metabolism of the nucleins of the body-cells (chiefly leucocytes) in response to the invasion of organic or inorganic toxins from the intestine, the gouty diathesis representing the inadequate power of the protective cells to successfully resist or prevent the absorption of the poisons.

Others give a prominent place to the liver in the diseased process, as first promulgated by Murchison and afterwards accepted by Yeo and in part by Luff, but Hutchinson is probably correct in regarding the part played by the liver as a negative one, the organ failing to perform its chief function as a destroyer of poisonous substances absorbed by the portal vein.

Duckworth, Laycock, and others still support the view advanced by Cullen that the disease is of neuro-humoral origin, the central nervous system playing an important *rôle* in the uric-acid production.

The views of Haig (who is a firm believer in the uric-acid origin of the disease as explaining all the symptoms of the acute

and chronic affection), that gout and rheumatism are the same preventible disease, caused by poisoning through flesh and tea consumption, are accepted by very few.

Falkenstein has started the theory that the uric-acid formation depends upon disease of the peptic gland-cells, which produce HCl; the only evidence he produces is the statement that he cures the gouty symptoms by large doses of the mineral acid. The theory is at total variance with the fact that gout does not occur as a complication in those diseases of the stomach where the HCl is demonstrated to be constantly absent.

Diagnosis.—A painful, acute joint-affection, especially if it be the great-toe articulation in a male patient past thirty-five years of age given to excesses in wine and eating, and especially if his family history is gouty, can scarcely prove to be anything else but gout. A history of previous attacks and the discovery of uratic deposits in other joints or in the rim of the ear will render the diagnosis certain.

Acute rheumatism is differentiated easily by the characteristic distribution of the joints involved, several corresponding articulations in two or more limbs being usually affected at the same time. The constitutional symptoms—severe fever and sweating—are present, and there is liability to cardiac complications and hyperpyrexia. In gout the local pain is out of all proportion to the febrile disturbance, which may indeed be absent, and in first attacks only one joint is usually affected; sweating is rare; the affected joint pits on pressure, and desquamation of the cuticle over it always occurs. Murchison, in his clinical teaching, always laid great stress on one diagnostic point in first attacks. In gout a large joint is never, or rarely ever, affected in a first seizure, whilst this is common in rheumatism. The evidence of age, sex, habits, history, and occupation are all valuable and, when taken together, conclusive.

Little importance need be attached to the state of the urine, as there is not the close relationship with the gouty paroxysm and the excretion of uric acid that is usually stated. The urine in both acute rheumatism and acute gout may present identical febrile and uric-acid characteristics. It is, however, different with the urine passed between the attacks, as often, but not by any means always, there is a diminished elimination of uric acid in gout; but this latter fact is useless in the diagnosis of first attacks. In rare doubtful cases a history of recent severe exposure to cold and wet may settle the diagnosis in favour of acute rheumatism.

Rheumatoid arthritis is often mistaken for chronic gout, and the old and still popular name of rheumatic gout is an evidence of the difficulty in differentiating the two affections. Rarely, if ever, do both diseases exist together; hence the

term is a misnomer. The chief diagnostic points have been already mentioned upon page 506. The sex, age, history, habits, and occupation should be considered, and the joints of the great toes and the rim of the ear should always be examined for tophi.

Prognosis.—The chief points bearing upon prognosis have been already incidentally referred to. The subjects of gout very often live to a good old age, as the attacks have but a slight tendency to shorten life; but the outlook is always serious in the *retrocedent* type of the disease. The prognosis as regards frequency and severity of the attacks will depend to a considerable extent upon the strength of the hereditary factor as disclosed in the family history, and upon the individual himself in as far as his will-power or grit will enable him to abstain from over-eating and wine-drinking.

The chief point, however, in prognosis is the presence of renal disease. Once there is evidence that structural alteration in the kidney has become established, with its secondary vascular and cardiac changes, the prognosis is grave, though many cases live for years after the kidneys become affected.

Treatment.—Upon pages 336–345 in the author's 'Dictionary of Treatment' the details and principles of the therapeutics of gout have been fully discussed; only the main points need be here repeated.

In the acute paroxysm the joint should be enveloped in absorbent wool and covered in with thin mackintosh-sheeting or oiled silk, and the patient confined to bed or a sofa. A mild saline, after the first severe onset has been modified by a dozen hours of rest, and a milk-diet are desirable. Colchicum is the only drug capable of controlling the gouty inflammation, and, notwithstanding the dangerous characters which tradition has often associated with its exhibition, if carefully administered it is quite safe. Thirty minims of the wine may be given as soon as the remedy is obtainable, and half this quantity may be administered every three or four hours. Salicylate of soda may be substituted for it if there be any peculiarity in the patient's condition to warrant its prohibition. Poulticing, leeching, and cold applications must be condemned, and, as a rule, the less morphine prescribed the better.

Between the gouty seizures the main indication is the correction of those obvious errors in the diet, drinking-habits, and sedentary life of the patient which have induced or aggravated the disease.

Diet.—The natural revolt against the rigid prohibition of animal food, which was based upon the old view that this was the main factor in the production of uric acid, has led to mistakes upon the other side by those who hold the newer view that uric acid is the result of the increased destruction

of the leucocytes. Hence it is not uncommon to see foods recommended which are rich even in nucleins and purin-bases, such as pancreas, thymus, red meats, game, and roe. It is, however, certain that if such are permitted they should be only taken in small quantities. Both extreme views should be avoided. The experiments of Schmoll and Reach show that the joint-inflammation is increased by sweetbreads and food rich in nuclein, and they believe that some of the exogenous purin-bodies are retained, since the increase in the excretion of uric acid is not in proportion to the amount of nuclein taken in.

The amount of the food swallowed is often of more importance than is its chemical constitution. The writer has frequently observed that when red meat is prohibited the patient takes to enormous quantities of farinaceous food to make up for the deficiency. Gormandising must be avoided, no matter how mild and apparently unobjectionable the quality of the food may be; and many gouty patients are unable to digest farinaceous stuffs. Fats, as cream and butter, may be taken freely, and salads are beneficial. Mineral-waters (aërated), or hot water flavoured with a little lemon, may be freely taken with the view of encouraging elimination.

Alcohol in every form should be prohibited as a routine beverage, though it is fashionable nowadays to prescribe a little old port or good champagne as a protest against old-fashioned treatment. If alcoholic stimulants are indicated by heart-failure or other complication, a little good whisky, well diluted with potash-water, is the best and safest form for the exhibition of a stimulant. Active open-air exercises should be pressed to the margin of fatigue. Smart walking, horse-riding, and golf may be recommended. Mineral-water springs are in great repute; at home, Bath, Buxton, and Strathpeffer being the best, whilst abroad Carlsbad is the favourite. Iodide of potassium, in combination with the bicarbonate in full doses, is of great value in chronic gout when made to alternate with short courses of salicylates, combined with small doses of colchicum. There is a reaction against active dosing with strong alkalies, lithium, piperazine, lysidine, and urotropin; all mineral waters containing much sodium should be avoided.

When symptoms of retrocedent or irregular gout manifest themselves, the best plan is to apply counter-irritation or hot-packs to the seat of the original joint-affection, and to trust to excretory treatment by alkaline diuretics and diaphoretics, only using colchicum with the greatest circumspection, or not at all.

Local treatment directed to the joints which have become the site of deposits must be cautiously employed. Blistering should be seldom resorted to; iodine preparations continuously

employed are unobjectionable. Massage, hydropathy, and passive movements often retard or prevent crippling. There is little to be said for the permanent effects of cataphoresis, though marked temporary relief is often obtainable from its employment.

GUINEA-WORM DISEASE

Also known as Dracontiasis. This is a common disease in the tropics, constituted by the presence of a worm, a member of the Filarida family—*Filaria* or *Dracunculus medinensis*—which is found in the subcutaneous and intermuscular connective tissue.

The young embryos penetrate the ventral joints of the fresh-water cyclops, and after these have been swallowed in drinking-water by man the parasite penetrates his alimentary canal and, reaching the subcutaneous tissues, the worm makes its way by burrowing head foremost in the direction of the feet. After reaching a point, usually in the vicinity of the foot and ankle, the worm may be found lying like a cord, one-tenth of an inch in diameter, under the skin, and measuring from one to six feet in length. Only the female worm is met with. Finally she penetrates the cutaneous covering and extrudes her millions of young active embryos, especially when the affected limb is placed in water. When all these have been evacuated through her mouth the worm begins to slowly part from her host through the small cutaneous opening. Her removal is usually facilitated by the patient gently winding her body round a minute piece of wood as thread is wound upon a reel or spool, a process which requires many hours or a few days. A speedier method of getting rid of this usually harmless guest is to inject a few drops of the B.P. liq. hydrarg. perchlor. into its track, when its death and subsequent absorption take place. Generally the parasite causes little, if any, irritation unless premature attempts be made at its dislodgment. Usually only one worm is found, but occasionally two or three may exist in the same host.

Other *filaria* are met with in addition to those described under 'Chyluria,' page 219. Of these *F. loa* is often found under the conjunctiva in the natives of the African West Coast; it measures over one inch in length. *F. labialis*, *F. hominis oris*, and *F. bronchialis* have been occasionally met with in the regions of the body indicated by their names.

HÆMATEMESIS

The term is applied to the vomiting of blood, though the source of the bleeding may not be the gastric vessels. The first step in the recognition of this sign is to exclude those conditions in which blood comes up through the mouth without having been in the stomach.

In hæmoptysis the blood is coughed up, but patients are often unable to discriminate between coughing or hawking up, and vomiting. The blood in hæmoptysis is usually bright red, alkaline and frothy from the presence of minute bubbles of air, and mucus may be mixed with it. When coming from the stomach it is usually darker in colour, acid in reaction from admixture of gastric juice, and food may be mixed with it. Should its source be from an artery of considerable size in the stomach walls, it may be a bright scarlet colour and copious in amount.

As a rule, however, it is more or less altered in its characters by a partial digestion, and in one of the most frequent forms coming under the physician's notice it presents the appearance of the grounds of coffee, the presence of blood not being even suspected by the patient. This is the usual state of the gastric hæmorrhage occurring in cancer of the stomach, and where the amount is small a careful examination may be necessary to detect its presence. The microscope will usually reveal red corpuscles; but in difficult cases, where these are much altered by the gastric juice, a small quantity of the filtered stomach-contents should be chemically tested.

This is done by evaporating a drachm of the filtrate to dryness in a small dish, placing the residue on a slide with a minute crystal of common salt and a drop of acetic acid, and gently heating after covering with a microscopic cover-glass, adding a little acid at the margin of the cover from time to time. Upon cooling, hæmin crystals will be found by using a power of about 350 diameters.

This test will eliminate those common sources of error arising from coloured articles of food, as strong coffee, red fruits, wines, &c.; and it will prevent mistakes being made in cases where the patient has been taking iron or bismuth, or where bile is present in the stomach. The presence of blood in the vomit is not to be taken as proof of gastric hæmorrhage until various sources of error have been eliminated. Thus the original source of the blood may have been an hæmoptysis, the blood being subsequently swallowed. Epistaxis, fracture of the base of the skull, bleeding from the mouth, pharynx, or œsophagus are all conditions in which the blood may find its way into the stomach, to be rejected by vomiting. A careful examination will usually reveal the nature of the case, but it may be difficult to detect the fraudulent methods resorted

to by hysterical subjects who feign hæmatemesis, unless by closely watching them in hospital. Where the blood comes from a malignant ulceration in the gullet, the accompanying symptoms of dysphagia usually prevent an error in diagnosis.

The varicose condition of the large veins at the lower end of the œsophagus which is so frequently found in cirrhosis of the liver may lead to rupture, and the subsequent evacuation of the outpoured dark blood by vomiting simulates a true gastric hæmorrhage so closely that a differential diagnosis may practically become impossible. The history, however, always throws light upon the hepatic condition, even when there are no liver-symptoms. Sometimes large quantities of dark venous blood well up and trickle from the mouth in these œsophageal varices in a manner which is highly characteristic.

Occasionally a fatal hæmatemesis follows the rupture of an aneurism into the œsophagus, and the writer once witnessed a similar event in cancer of the gullet which had ulcerated into the aorta.

Hæmatemesis arising from true gastric hæmorrhage may be caused by the following : (1) *Ulcer* ; (2) *Cancer* ; (3) *Congestion* of the mucous membrane, the result of portal obstruction from hepatic or splenic disease, or from the mechanical stagnation caused by mitral lesions ; (4) *Injuries*, as blows upon the epigastrium ; (5) *Poisons* of the corrosive class, and phosphorus, alcohol, and other irritants ; (6) *Blood-conditions*, as seen in anæmia, leukæmia, scurvy, purpura hæmorrhagica, hæmophilia, icterus gravis or acute yellow atrophy, yellow fever, malaria, typhus and typhoid fevers, diphtheria, pyæmia, cholæmia, Bright's disease, &c. ; (7) *Aneurisms* of the splenic, hepatic, or gastric arteries ; (8) *Gastritis* of the acute toxic variety may, though rarely, produce hæmatemesis ; (9) *Vicarious menstruation*, which is so rare as to be considered a very doubtful cause, unless ulceration is also present ; (10) *Duodenal ulcer*, though mentioned last, must never be omitted from consideration. Melæna may accompany any of the above varieties of gastric hæmorrhage, but it is chiefly in the last-mentioned that its presence is of diagnostic importance.

The Diagnosis, Prognosis, and Treatment of these different causes of gastric hæmorrhage are detailed under their respective headings.

HÆMATOPORPHYRINURIA

This is characterised by the presence of a purplish-red urine containing hæmatin devoid of iron. The condition has been found in the great majority of cases to follow the prolonged administration of sulphonal, *especially in women*. Trional and tetronal rarely lead to its appearance. The hæmatoporphyrin is derived from the blood-pigment and usually is

associated in the urine with other pigments. Though death has been known to follow this condition in a few cases, it is of little clinical importance, since it is often present in appreciable amount in various diseased conditions, as in intestinal hæmorrhage, Graves' and Addison's diseases, pleuritis with effusion, phthisis, cancer, typhoid fever, &c. The guaiacum-test does not reveal its presence, which is best demonstrated by the spectroscope, though the addition of acid or alkali may be needed to avoid fallacies.

T. K. Monro states that the condition sometimes occurs in paroxysmal form, and he believes that the coloration is due mainly, if not solely, to other pigments than hæmatoporphyrin.

When following sulphonal administration, this drug should be immediately suspended, and Müller recommends the free use of alkalies.

HÆMATURIA

The appearance of blood in the urine is a symptom met with in many medical and surgical affections ; with the latter the present brief epitome does not deal, save in so far as the differential diagnosis from medical lesions is concerned.

When the amount of blood mixed up in the urine is trifling the condition is liable to be overlooked, but it is remarkable how small is the quantity which can be detected by the trained eye. Thus one drop of blood will give a smoky tint to a wineglassful of urine.

The colour of the urine may vary from the palest shade of pink to that of strong porter. In the former case its smoky tint may resemble a weak beef-tea, and in the latter it may even approach to the blackness of ink ; between the two extremes all shades of red may be noticed. In acid urine the colour is often dark red, whilst in alkaline urine it is usually bright red.

It must be remembered that various drugs administered to the patient may stain the urine, as sulphonal, salicylates, logwood, rosaniline, carbolic acid, senna, and rhubarb ; fruits like bilberries, and beetroot, may produce the same effect. Albumin is invariably present when blood is mixed with the urine.

In doubtful cases various tests may be employed, the most obvious being the microscope. The red cells in acid urine may be easily detected ; though their margins are often crenated in concentrated urine, they are otherwise usually little altered, but rouleaux are rarely seen. In alkaline states and in dilute urine the discs swell and soon lose their shape, and when alkaline fermentation sets in they disappear.

A small quantity of the suspected urine, on being treated by the method mentioned upon page 517, will show Teichmann's hæmin crystals.

The guaiacum-test is easily applied by mixing a little of the fresh alcoholic tincture with the same amount of ozonic ether in a test-tube and gently pouring the urine upon it, when a blue colour strikes where the two liquids come in contact. This indicates the presence of *blood-pigment*, but iodides will also produce the same reaction. The spectroscope will give the blood-spectrum recognisable by the hæmoglobin bands between the D and E lines in the yellow and green.

The source of the blood may be the kidney-substance, its pelvis, or ureter, the bladder, or urethra.

Renal Hæmaturia.—When the blood comes from the kidney it is intimately mixed with the urine and clots are not observed, unless very small ones. The causes of renal hæmorrhage are numerous ; the most important are : (1) *Acute Bright's disease*, or a sub-acute attack upon the top of the chronic form of the disease. (2) *Congestion* of the kidney, caused by sudden chilling of the entire cutaneous surface or by the administration of cantharides, turpentine, potassium nitrate, copaiba, carbolic acid, &c., or by the mechanical stagnation of the blood, the result of mitral lesions or portal obstruction. (3) *Blood-conditions*, as purpura, hæmophilia, scurvy, leukæmia ; various fevers, as malignant variola, typhus, typhoid, and malaria. (4) *Parasites*, as in the endemic forms caused by *Bilharzia hæmatobia* and *Filaria sanguinis hominis*, and hydatids. (5) *Renal infarction*, as in the arterial pyæmia of malignant endocarditis, and very rarely aneurism of the renal artery or thrombosis of the renal vein. (6) *Renal calculus.* (7) *Tumours.* (8) *Tuberculosis.* (9) *Injuries.* (10) *Floating kidney.* (11) The so-called ' Renal Epistaxis,' in which no cause or lesion can be detected.

Diagnosis.—The hæmaturia of Bright's disease is recognised by the anasarca, casts, and other signs or symptoms of the disease. Acute congestion of the kidney is usually differentiated by its history—the appearance of blood *immediately* following a profound chill. In chronic cases the presence of valvular disease, or other cause of venous stagnation or portal obstruction or liver-disease, easily settles the diagnosis ; the amount of blood corresponds in these cases to the quantity of albumin always present.

The bleeding caused by renal irritants is easily discriminated by an examination of the history.

The hæmaturia caused by altered states of the blood is always associated with the specific signs and symptoms of the primary disease, and hæmorrhages from other mucous surfaces and into the skin are as a rule present.

Filariasis and Bilharziosis are tropical or sub-tropical in their distribution, and are recognised by the presence of the ova or embryos in the urine. Psorospermiasis, caused by psorosperms belonging to the protozoa, have been in a few

cases identified in the urine in hæmaturia, and the cysts or portions of the membranes from renal hydatid may be also found in the hæmaturia produced by the echinococcus.

Renal infarction may in rare cases be the first sign of the blocking caused by the emboli of malignant endocarditis, in which case the diagnosis is difficult; but the presence of the cause which has led to the cardiac infection will always be found if carefully searched for. (See under 'Endocarditis,' p. 375.)

The hæmorrhage from *renal calculus* is usually intermittent, with long or irregular intervals; the source of the blood is not the renal parenchyma—it comes from the pelvis of the kidney. It is liable to follow exertion, is associated often with pain, and usually stops when the patient remains at rest in bed. It is seldom profuse; and though the blood is well mixed with the urine it is not so intimately blended with it as in Bright's disease, so that the corpuscles fall to the bottom of the urine-glass and may be easily recognised from the clear and bright urine after this has been permitted to stand for a few hours. Palpation of the flank may give valuable aid in diagnosis, and the use of the cystoscope may demonstrate the blood flowing from the orifice of the ureter upon the affected side. Catheterisation of the ureter is difficult except in the female, and it may mislead by the presence of blood caused by the instrument itself. In the intervals between the attacks of hæmaturia the blood is free from albumin, but in chronic cases pus may be present constantly, which will cause slight albuminuria. Fragments of the concretion may sometimes be found in the urine, which will indicate its nature.

Tumours, or new-growths, often give rise to profuse hæmorrhage, which is of a more constant type than that produced by stone; pain may be absent, and the bleeding often occurs when the patient is resting in his bed. The growth is generally palpable, and is as a rule easily differentiated from the distension caused sometimes by a stone blocking the ureter, as this is not associated usually with hæmorrhage. Fragments of the neoplasm may occasionally be found in the urine and identified by the microscope.

Tubercular disease of the kidney may first reveal its presence by hæmaturia, and the diagnosis can then only be made by the detection of tubercle in some other part of the body, notably in the testicles or vesiculæ seminales. Exertion has little or no influence upon its recurrence; the amount is very slight usually, and there may be long intervals between the attacks. As in stone and tumour, there may be small worm-like clots which have formed in the ureter; and when one of these is expelled after a temporary cessation of the hæmaturia, and the latter immediately returns, as pointed out by Newman,

the renal source of the hæmorrhage is demonstrated. Tubercle bacilli may be found in the urine by the microscope, or their presence may be detected by culture-experiments or by inoculation of the guinea-pig with the fresh urinary sediment. In one very interesting case of renal tuberculosis under the writer's care the diagnosis was demonstrated by Lorrain Smith, who was able to stain the bacilli *in situ* in the interior of a hyaline cast of one of the uriniferous tubes. In this case the disease had originated in the testicle, which had been removed by Professor T. Sinclair. Often pus is present in the urine, and the amount of albumin is always much greater than the quantity of blood and pus will account for—which is a valuable diagnostic point.

The renal hæmorrhage caused by injury, as blows or falls from a height, is easily recognised unless there be injury to the lower part of the urinary tract at the same time. When a movable or floating kidney is the source of the bleeding, the other signs and symptoms usually leave no doubt about the diagnosis.

The cases in which no lesion has been detected in the genito-urinary tract are generally beyond diagnosis. The presence of marked hysterical symptoms in the patient should arouse suspicions of this condition after all other sources of hæmaturia have been eliminated; and in one such case of the writer's, in a highly hysterical and neurotic girl, dilatation of the urethra and exploration of the bladder gave no clue to the source of the hæmorrhage, but a cure followed the operation. A similar result has followed exploration of the kidney.

Blood coming from the bladder or urethra is of surgical interest only, but it is necessary for the physician to distinguish it from a hæmaturia of renal origin. The usual causes are stone, tumour, cancerous or tubercular ulceration, parasites, prostatic disease, and affections of the posterior urethra which permit of a back-flow of the blood into the bladder.

The urine in vesical hæmorrhage usually contains much mucus, and often pus, and the blood is frequently seen to be in clots of such a size as precludes the probability of a kidney or ureter origin. Usually the hæmorrhage is bright and not intimately mixed with the urine. If the patient during rest in bed passes his urine under the physician's observation, the first passed may be normal in appearance; but as the stream continues to flow the urine assumes a red colour, and towards the end of the act pure blood comes. An examination of the sediment will show the absence of renal cells and casts and the absence of the characteristic epithelium from the pelvis or ureter, whilst it will reveal the broad vesical cells mixed with much mucus, pus, and blood. Washing out the bladder sometimes gives aid in the diagnosis, as when

the bleeding is from vesical ulceration the colourless fluid injected continues to be blood-stained all through the operation, whilst in renal hæmorrhage the staining practically stops as soon as the first flushing is completed.

The cystoscope, after the washing-out of the organ, may reveal the bleeding-point or may show the normal urine trickling from the healthy orifice of each ureter. Catheterisation of the ureters is satisfactory in skilled hands.

Blood from a urethral source is easily recognised when it comes away independently of micturition, but sometimes when the urethra is affected near to the neck of the bladder the blood flows back into the organ and simulates vesical hæmorrhage. In other cases, when the hæmorrhage is small, the blood may remain in the urethra until micturition, when it is expelled almost pure at the very beginning of the act, the urine being normal. A similar state of affairs may exist when the source of the bleeding is prostatic.

HÆMOGLOBINURIA

This is a condition characterised by the presence in the urine of blood-pigment, either in the form of hæmoglobin or its oxidation-product—methæmoglobin.

Under normal conditions the breaking up of the blood-corpuscles must always be followed by some slight degree of hæmoglobinæmia, but the organism is able to dispose of the *débris*. In diseased conditions the hæmolysis is extensive, and the *débris* or useless pigment either in the form of oxy-hæmoglobin or methæmoglobin produces a marked degree of hæmoglobinæmia (*i.e.* the blood-serum is charged with the dissolved pigment) which upon reaching the kidney is eliminated in the urine. No red blood-corpuscles are found in the urine, the pigment only being excreted, which gives this fluid a deep red or reddish-brown tinge, and in some instances a jet-black colour, as described under ' Blackwater Fever,' p. 107. There is a copious deposit of dark-brown sediment, which is found to consist of granules of pigment, and small fragments of red discs are sometimes recognisable mixed with urates. Casts are often seen containing the granular hæmoglobin *débris*. When red discs are found, which is rarely, their presence is accidental, being due to the renal irritation caused by the abnormal activity of the organ in its eliminatory efforts. The urine is usually acid and of lowered s.g., and always contains albumin, which may continue for some time after the attack has passed off ; sometimes slight albuminuria has been found to precede the paroxysm. The albumin is not serum-albumin, but serum-globulin. The urea is usually much increased and the urine, clear when passed. soon becomes

turbid. The spectroscope reveals the presence of oxyhæmoglobin and methæmoglobin after filtrating and after dilution, if the sample be very dark in colour. The former is recognised by its two bands in the yellow and green, near D and E, and the latter by two paler bands nearly in the same position, with a third band, which is characteristic, in the red near C. One or other pigment may only be present, but usually both are found together.

Hæmoglobinuria is met with in several types—*Toxic*, *Febrile*, and *Paroxysmal*—but probably all are of toxic origin. Toxic hæmoglobinuria is caused by several poisons which possess the power of causing rapid destruction of the red corpuscles ; the most common of these hæmolytic poisons is potassium or sodium chlorate. Arseniuretted hydrogen, carbolic acid, pyrogallic acid, carbon monoxide, phosphorus, naphthol, sulphuretted hydrogen, quinine, hydrochloric and sulphuric acids, and toluylendiamin all possess hæmolytic properties when administered in poisonous doses. Of the same type is the hæmoglobinuria which results when the blood of sheep and other animals is transfused into the human veins. The *epidemic infantile hæmoglobinuria* of Winckel is evidently the result of some organic poison not yet identified, which also produces jaundice, cyanosis, collapse with marked fall of the body-temperature, and fatal coma.

Febrile hæmoglobinuria is sometimes met with as a result of the action of the poisons of typhus, typhoid, rheumatic, yellow, and scarlet fevers. It is not unusual in severe malarial fever, and is the chief clinical feature in blackwater fever ; it is also met with in pyæmic states, in syphilis, though rarely, and after severe burns. It is also believed in rare cases to follow severe exposure to heat and cold—a not uncommon sequence in horses.

PAROXYSMAL HÆMOGLOBINURIA

This is a disease which manifests its presence by attacks at irregular intervals, usually commencing with chills, followed by the voiding of red or dark-coloured urine containing oxyhæmoglobin, or more commonly methæmoglobin without red corpuscles.

Etiology.—This rare affection is met with usually in young male adults ; a few cases have been recorded in children and women.

Exposure to cold is usually the exciting cause of the attacks in individuals who have suffered before, and paroxysms may occur only in the winter in many patients who have suffered for years. Prolonged exertion and mental over-work are also exciting causes.

Syphilis and malaria are supposed to be powerful factors in the causation of the disease ; Copeman states that a definite

specific history was obtainable in all his cases. In the only typical case seen by the writer, both these affections were easily excluded. Rheumatism is supposed to be a predisposing cause, and so also is gout.

Raynaud's disease seems closely associated with or related to hæmoglobinuria, many cases of the affection showing attacks of red-coloured urine along with the symptoms of deadness of the fingers and toes or other signs of local asphyxia. The associated symptoms are always produced by exposure to cold or dipping the extremities into cold water. Many cases, however, of Raynaud's disease never show symptoms of hæmoglobinuria.

Symptoms. — The attack is usually ushered in by a feeling of chilliness, often by a distinct rigor which follows the exposure to cold. Though the patient shivers and may complain of a dead numbness in his hands and feet, there is often no elevation of temperature, and in many cases the temperature is markedly sub-normal ; but some hours later, after the passage of the pigmented urine, there is usually a rise of several degrees. Vomiting and lumbar pain are sometimes noticed, but often, beyond a feeling of lassitude which soon passes off, there may be no symptom of illness save the altered character of the urine. This may be voided within an hour after the first symptoms appear ; more frequently it follows after two or three hours, and the entire attack is over in five or six hours.

The urine presents the characters already described upon page 523, and the colour varies considerably ; in the typical case already referred to it was always of a bright scarlet colour, as if some red ink had been mixed with it. When the urine remains long in the bladder the oxyhæmoglobin is changed by the acidity of the secretion into methæmoglobin, and sometimes into acid hæmatin (Copeman).

The attacks, as already stated, come on at very irregular intervals ; there may be two or three within twenty-four hours with quite normal or only slightly albuminous urine voided between them ; or there may not be an attack for many months till a smart exposure precipitates a fresh paroxysm.

Anæmia and an icteroid tinge may remain after the severe hæmolysis, and occasionally jaundice follows.

Ralfe has recorded a number of cases in which the attacks alternated with paroxysms of albuminuria, and he regarded the latter as an indication of a mild hæmoglobinuria in which the pigment had been used up in the organism without appearing in the urine. As already stated, the origin of the albumin is the globulin : it is not owing to the presence of serum-albumin.

The attacks are never fatal, but the anæmia which they induce may prove serious if neglected.

Pathology.—Little is known beyond the fact that the hæmoglobinuria is the result of a hæmoglobinæmia caused by increased hæmolysis. The origin of the latter is obscure, and it is usually attributed to some inherent weakness in the blood-forming organs, which produce red cells that are easily broken up by some toxic agent operating in conjunction with cold or muscular exhaustion. The fact that the blood-serum immediately before or during the attack is charged with the colouring-matter of the dissolved or disintegrated corpuscles in the vessels shows that there is no reason to suppose that the origin of the disease is renal. The serum of a blister applied at the beginning of an attack always affords evidence of the presence of hæmoglobin on examination by the spectroscope. The blood-count at the beginning of the paroxysm always shows a marked reduction in the number of red cells. Ehrlich reports the presence of decolorised red corpuscles, also an increase in the platelets; rouleaux do not form.

Recently Eason has discovered a toxic substance or hæmolysin present in the blood, which under the influence of cold fixes itself to the red blood-cells; but not till the blood becomes warm again by circulating in the deep parts does hæmolysis occur. Bradford claims that these facts explain the entire phenomena of the disease.

Diagnosis.—The only diseased condition liable to be mistaken for hæmoglobinuria is *hæmaturia*, but the absence of red blood-cells in the urine, which contains not blood, but only blood-pigment, is conclusive when taken in conjunction with the spectroscopic characters. The old name 'hæmatinuria' is a misnomer, the colouring-matter not being hæmatin.

Prognosis.—The disease seldom ends fatally and many cases show a spontaneous tendency towards recovery. The prognosis mainly depends upon the patient's ability to protect himself from exposure during the cold months; the disease usually disappears upon his removal to a hot or warm equable climate.

Treatment.—Rest in bed is essential, and protection from cold is the chief preventive and curative agent, removal to a warm climate affording the best prospect of final recovery. Murri and many other authorities recommend iodides in large doses between the attacks, because of the common relationship believed to exist between the disease and syphilis. Arsenic and quinine have their advocates, probably on the grounds of a malarial theory of its origin. Quinine is still believed to be the cause of malarial hæmoglobinuria by some. Tomaselli has recorded several cases where quinine brought on the symptoms of hæmoglobinuria in patients who had previously suffered from malaria; and some authorities maintain that quinine never produces a paroxysm unless the patient has already suffered from an invasion of the malarial parasite.

Nitrite of amyl was found by Chvostek to cut short the paroxysm if administered at the outset. Iron is always indicated in the anæmia which follows severe attacks.

HÆMOPHILIA

This is a congenital hereditary disease or diathesis characterised by a tendency to spontaneous bleedings and to extensive hæmorrhages on slight provocation, and to joint-affection.

Etiology.—Heredity is the only known etiological factor, combined with sex. The disease is almost exclusively confined to males, and is as a rule transmitted by healthy members of the female line, who pass the disease to their male offspring, the girls escaping; but these in turn transmit the affection to their male children. The sons of male bleeders do not suffer, and consanguinity is not a factor in the propagation of the diathesis, which is not confined to race, geographical or class distribution.

Pathological Anatomy.—This only reveals the anæmia or extravasations of blood which have recently preceded death. No definite changes in the chemical composition of the blood or in the histology of the vessels are observable.

The joints may be found altered by the successive hæmorrhages which have occurred in their interior, the final changes ending in thinning and fibrillation of the cartilages, closely resembling the condition met with in rheumatoid arthritis.

Of the *pathology* of the disease nothing is known. Diminished coagulability of the blood is supposed by many to be the explanation of the disease, and Wright reports that the period at which coagulation should occur is markedly delayed. In many instances, at the beginning of a hæmorrhage the blood is seen to coagulate as if it were in every way normal blood.

Symptoms.—These appear usually within the first year of life, and in a considerable number of cases have been noticed to follow the operation of circumcision performed at the end of the first week. In many instances the first signs of the diathesis have not shown themselves till the child has reached the age of seven or ten years, or even later in rare cases. The subjects of hæmophilia, or 'bleeders' as they are usually termed, are apparently quite healthy, and in the absence of a history no suspicion of the diathesis is aroused till an extensive and uncontrollable hæmorrhage follows some slight wound. The bleeding is usually of the character of a capillary oozing, and an enormous quantity of blood may be lost from a trivial wound, which continues to bleed for days or weeks. Thus death has frequently been caused by the extraction of a tooth and by the operation of scarifying

the gums, or even by the minute incisions made by the lancet in vaccination or from the bite of a leech.

Spontaneous hæmorrhages are usual, and the most frequent of these is epistaxis, which is answerable for the largest number of deaths in hæmophilia. Bleeding from the gums is also very common, especially in very young subjects; and after the age of ten or twelve years spontaneous hæmorrhages from the bowel, stomach, bronchial surface, kidney, or urethra are noticeable. Bleeding into the serous cavities is much less common.

Interstitial hæmorrhages also occur spontaneously or from blows: the blood-tumours thus formed may assume large dimensions, and if incised are almost always fatal; petechiæ or ecchymoses appear upon the most trivial provocation.

Death may occur from traumatic or spontaneous hæmorrhage, in spite of all efforts for its arrest, within twenty-four hours or earlier; but except in the cases of infants it is usually at a much later period. After profound loss of blood, when the patient is apparently almost lifeless and pulseless, recovery may slowly set in, and the convalescence is always protracted.

Joint-hæmorrhages, or hæmarthroses, usually do not begin to show themselves till after the fourth or fifth year of life. The large joints, like the knee, ankle, and shoulder, suffer most frequently. They become suddenly swollen, tender, and painful, and fever often accompanies the extravasation of blood into the joint-cavity. Gradually, recovery is complete in a few weeks after first attacks, but as the hæmorrhages recur the joints become more or less destroyed, anchylosed, and useless.

Diagnosis.—This is usually easy, especially as an hereditary history is obtainable in the majority of cases. *The hæmorrhages met with in new-born children* are often mistaken for evidences of hæmophilia, and chief amongst these is the condition in which severe and sometimes fatal umbilical bleeding occurs upon the separation of the cord. The points to be observed in the differential diagnosis are that children of either sex are equally affected; jaundice is very often present; no hereditary history of hæmophilia is usually obtainable, and when the infant recovers there are no signs of hæmophilia afterwards to be observed.

Melæna neonatorum shows itself by the second or third day, and is the result of a gastro-intestinal hæmorrhage; as in the preceding condition, girls are as frequently affected as boys, and usually there is no history of heredity.

Scurvy in the infant is recognised by the presence of periosteal swelling and tenderness and sponginess of the gums, the hæmorrhages under the skin and from the kidney being but an accompaniment when present.

Purpura may be mistaken for hæmophilia where there are many cutaneous or deep hæmorrhages, epistaxis, &c.; and in

it there may be joint-troubles from bleedings into the interior of the articulations. The diagnosis in such cases must depend upon the absence of the history of the congenital and hereditary hæmophilia, and the evidence that the purpura is but a new condition supervening in a patient who never before had presented hæmorrhagic tendencies. Hæmophilia, in other words, is a *permanent* condition, and the bleedings in scurvy, purpura, leukæmia, &c., represent a transient blood-change.

Prognosis.—This is very grave when hæmorrhages occur in infants and very young subjects who bear bleeding badly. If the bleeder be tided over the first fifteen years of life his chances brighten; and though the tendency to bleed on slight provocation does not, as some writers affirm, pass away after middle-life, the subjects of hæmophilia as they approach that period bear their losses better and may in some instances live to a fair age. Legg, who has watched many bleeders for years, states that middle-age is within the reach of many of them.

Treatment.—Nothing can be done to alter the congenital condition, but the greatest care is necessary in protecting the bleeder from injuries of every kind. The only permissible operation is that of vaccination, which must be performed with great care, as death has been known to follow. The general health must be scrupulously attended to.

Hæmorrhage must be treated upon the ordinary principles, but any attempt at the ligature of bleeding vessels is certain to prove fatal. Styptics are generally useless; and those which destroy the tissues, as the actual cautery and strong perchloride of iron, must not be employed. Cold, by means of ice or ethyl chloride, may be employed; adrenalin is recommended, but the writer does not hesitate to advise the application of the puff-ball (lycoperdon). Pressure is the most reliable agent, and it can in many cases be applied over a pad of the fungus laid upon the bleeding part, pushed into the socket from which a tooth has been extracted, or plugged into the nasal cavities in epistaxis.

Internally every known remote hæmostatic has been employed, but the only one apparently of any real value is the chloride of calcium in full doses. It is probable that the best result will be obtained by a saturation of the blood with the calcium salt for a period of four or five days at a time. Yeast and nucleinic acid are suggested upon theoretical grounds. Harkin maintained that chlorate of potassium, given with tincture of perchloride of iron, had almost specific virtues. Gelatin, albumin, thyroid-extract, and adrenalin have their advocates. Transfusion of blood or saline solution is resorted to in desperate cases, but it would seem much wiser to administer saline solution hypodermically by means of a fine needle, and not to risk the opening of a vein in those cases of spontaneous hæmorrhage which appear about to destroy life.

The treatment of the joint-hæmorrhage is to be carried out on recognised surgical lines—absolute rest to the part, elevation of the limb, a moderate degree of compression by skilful bandaging, and the local application of ice. *Puncture* of the joint has been advocated, but *incision* is universally condemned.

It is almost needless to say that the subjects of hæmophilia should be personally warned of their danger as soon as they come to an age capable of understanding their peril, and the marriage of the girls in the family of bleeders should be strongly discountenanced.

HÆMOPTYSIS

The term is synonymous with the popular one—spitting of blood—and includes all those cases where blood is expectorated from the bronchial, tracheal, or laryngeal surface and from the pulmonary vessels, as well as those in which an aortic aneurism or a cancer may ulcerate into the lower respiratory tract.

The source of the blood in hæmoptysis is usually the pulmonary vessels, and as the hæmorrhage finds its way into the smaller bronchial tubes it is brought up by coughing. The old term *bronchorrhagia* implied that the origin was from the mucous membrane, and it was employed in contradistinction to *pneumorrhagia*, in which the blood escaped from the branches of the pulmonary artery.

The bleeding may be of the nature of a capillary oozing, but more frequently it is the result of an erosion or ulceration of a pulmonary vessel, or it may be caused by the rupture of the small or miliary aneurisms constantly found in phthisis on the branches of the pulmonary artery.

The following are the conditions in which hæmoptysis is usually met with :

1. *Pulmonary tuberculosis.*—This is the cause of blood-spitting in the great majority of all cases coming under the notice of the physician. The subject will be dealt with in association with the other signs and symptoms of phthisis, but occurring as a single or isolated symptom it should always arouse suspicion. It may occur at any stage of the disease. When appearing early, before any other symptom, the older view that it was the *cause* of the lung-disease which followed is now practically abandoned for the doctrine that the bleeding is the *effect* of the tubercular lesion which is already present.

The amount of hæmorrhage in this early stage is usually small, and is probably caused by some hyperæmic or congestive state of the pulmonary tissues in the immediate vicinity of the tubercular deposit. At a later period the bleeding is caused by the ulceration of a branch of the pulmonary artery

whose coats have become infiltrated by the tubercular bacilli. More frequently, however, and especially in the fatal cases of hæmorrhage occurring in pulmonary phthisis, the coats of the infiltrated artery bulge towards the side on which there is the minimum of support, and a small usually sacculated aneurism forms, which projects into the tubercular cavity. The rupture of these dilatations produces copious hæmorrhages which may rapidly cause death.

2. *Pulmonary diseases other than tubercle*, as pneumonia, cancer, emphysema, hydatids, actinomycosis, gangrene, abscess, cirrhosis, bronchiectasis, and, though rarely, bronchitis, all more or less frequently cause hæmoptysis. To this category also belongs the so-called *endemic hæmoptysis*, met with in Japan and Formosa, which is caused by the presence of the *Distomum pulmonale*, found in numerous burrows scattered throughout the pulmonary tissue and small tubes.

3. *Valvular diseases of the heart*, especially mitral stenosis, cause bleeding by rupture of the pulmonary capillaries, which become distended from the obstruction to the return of the blood to the left ventricle. Of the same nature is the bleeding which occurs in active hyperæmia of the lungs caused by violent exertion, and in the passive congestions produced by the pressure of thoracic cancerous tumours or enlarged bronchial glands.

4. *Aneurism of the aorta* may suddenly burst into the pulmonary substance or into a bronchus or into the trachea, and cause a rapidly fatal hæmoptysis. Very often in such cases there are preliminary small bleedings occurring some time before the final rupture which are of great diagnostic value.

5. *Pulmonary apoplexy*, or hæmorrhagic infarction, is the result of an embolus or thrombus coming from the right side of the heart or from a large peripheral vein, which causes plugging of the pulmonary artery, as described under 'Embolism' and 'Thrombosis.' The large wedge-shaped infarcts so formed in the peripheral parts of the lung may be the source of characteristic hæmoptysis. They are prone to occur in mitral stenosis.

6. *Ulceration* of the mucous membrane of the larynx, trachea, or large bronchi, usually the result of syphilis, tuberculosis, or cancer, may cause extensive hæmoptysis.

7 *Morbid states of the blood*, as in purpura, scurvy, leukæmia, malignant types of fever, and in hæmophilia, are prone to induce bleeding from the mucous membrane of the respiratory tract. Of the same nature are probably the harmless recurring hæmoptyses described by Clark, who noticed their association with arthritic and gouty affections in elderly subjects.

8. *Menstrual suppression* has been credited with causing a *vicarious* hæmoptysis, but genuine examples of the

condition are so rare that only a few observers have met with any of them. This form of vicarious menstruation has also been noticed after double ovariotomy.

9. *Trauma*, especially any injury which causes fracture of a rib whose broken extremity pierces the pleura and lung, may lead to severe hæmorrhage. Injuries which suddenly compress the thorax violently, without fracturing ribs, may cause severe hæmoptysis. Caisson-disease is often complicated with hæmoptysis; and any injury, surgical or accidental, which causes a breach of continuity in any part of the respiratory tract or in the lung-tissue will give rise to hæmoptysis.

10. After all these have been excluded there remains a group of cases in which hæmoptysis appears suddenly in healthy young adults without any known cause, and the subsequent history shows that tubercle has never developed. The condition might be termed pulmonary epistaxis. The writer has had one patient under observation for thirty years, who had severe and repeated attacks of hæmoptysis when in about his twentieth year; such cases are comparatively common.

Symptoms. — As a rule, the onset is sudden. In many cases the first sensation of the patient is a saline taste in the mouth, followed by a tickling in the pharynx which produces coughing with the expectoration of blood. This is usually bright red, alkaline, and frothy from the admixture of fine bubbles of air, and mucus is usually present. The amount varies from a few sputa to pints; and in this latter case the blood may pour out of the nostrils, and may be mixed with clots. If examined carefully, the clots may be found to be the result of coagulation in the bronchial tubes, and may be distinguished from the larger masses which have formed in phthisical cavities in the lung-substance, and which are often mixed with pus.

The attack may last for hours or days. Usually blood-stained sputum is coughed up for several days after the first symptoms of hæmorrhage have abated, and often in these cases small sputa of dark blood are expectorated which have obviously been lying in the tubes for a considerable period. During the first part of the attack blood may be swallowed, and when this is evacuated by emesis it may be almost black, acid in reaction, and mixed with food; melæna often is noticed a few days afterwards, when the blood has passed into the intestines. When the hæmorrhage has occurred into a large cavity in the lung it may be very dark in colour when first pumped up, and occasionally death has resulted from shock without any of the blood being evacuated. The first effect of the hæmorrhage may be a profound shock which affects the heart, causing faintness; this, however, seldom ends in syncope, the pulse is very rapid and weak, the

temperature drops below the normal, and there are usually sweats and coldness of the extremities. These symptoms generally pass off as the dread and fright subside. In a considerable percentage of the cases the temperature rises above the normal upon the second or third day, probably from the presence of septic blood lying in the tubes or in cavities in the lung. When the hæmorrhage has come from an aortic aneurism or from a large sacculated dilatation on a vessel in the wall of a tubercular cavity, death may occur rapidly from loss of blood or from asphyxia, caused by the blocking of the respiratory tract. Such an event is, however, rare, and patients seldom die from the immediate results of hæmoptysis.

Diagnosis.—It is often difficult during the attack to ascertain the cause or source of the hæmorrhage; the first step in diagnosis is to exclude *hæmatemesis*, which is usually easily done, as the blood in this case is acid in reaction, mixed with food, generally dark in colour, and the patient is able to state that nausea and vomiting were associated with the attack. Moreover, it is brought up during a single effort and not in repeated mouthfuls, and a history of gastric pain or of previous unmistakable hæmatemesis is usually obtainable.

Epistaxis and hæmorrhages from the tongue, mouth, or throat are eliminated by a careful examination of the parts and an inquiry about the immediately antecedent symptoms. A varicose lingual tonsil has been found by Kinnicut to be a cause of hæmoptysis.

Malingering is not rare in hysterical girls, and must be guarded against; there cannot be any difficulty if the case is under observation during the attack. When small quantities of blood have been expectorated under suspicious circumstances, a careful examination of the gums should be made, when marks of injuries may be discovered. If the patient be closely watched, she may be detected biting the cheek or sucking her gums in order to cause bleeding. The pale pink blood-stained expectoration will reveal no ciliated or cylindrical cells, elastic fibres, or tubercle bacilli.

Laryngeal ulceration, either tubercular, malignant, or syphilitic, can scarcely cause a difficulty, as it is an extremely rare event for the first symptom of these conditions to be ushered in with hæmorrhage, so that there is always the history of previous laryngeal symptoms—hoarseness, aphonia, pain in swallowing, &c., to guide the physician.

The hæmoptysis being obviously the result of a hæmorrhage from the lung or bronchi, the next step is to determine its cause, and this, in the great majority of cases, will be found to be pulmonary tuberculosis. In the absence of evidence of other causes, as heart and non-tubercular lung diseases, the safest practice will be to assume that the cause is tubercle, until time proves this to be wrong. The characters of the

hæmorrhage in the different stages of phthisis have been already described. If the physical signs and symptoms of tubercular disease of the lung have preceded the hæmorrhage, there should hardly be a doubt in the case. It must always be remembered that the usual methods of examining the chest are not admissible during hæmoptysis ; thus percussion, deep breathing, and coughing are liable to aggravate the attack, or bring it on again if it has already ceased. Moreover, the bubbling of blood lying in the small tubes is liable to produce signs which may be misleading.

A careful examination of the mucoid or muco-purulent portions of the sputum is always available, and the presence of tubercle-bacilli is conclusive ; these may be found in the small dark clots which are coughed up towards the end of the attack on the second or third day, even in cases where there may be little or no mucus expelled. The presence of elastic fibres is also almost conclusive if gangrene and abscess can be excluded. The value of the thermometer should not be forgotten, as the temperature is usually elevated in tubercular cases, except during the first six or twelve hours of a smart hæmorrhage.

The hæmoptysis produced by other lung-diseases presents more or less characteristic features, even as regards the sputum, which assist in the diagnosis. Thus in cancer of the lung, in addition to the wasting and dyspnœa which have preceded the attack, the sputum will be stained a bright red, like the juice or jelly made from red currants. In gangrene the odour is pathognomonic, and the colour often resembles a tobacco-spit. In pneumonia the rusty sputum is highly characteristic, though often the prune-juice type may be present all through the disease. In pulmonary infarction the physical signs of the primary affection are in evidence, and at a late stage hæmatoidin crystals may be found in the brown sputum, but these are present in all old-standing hæmorrhages, as in abscess and empyema ; when the infarct is large, dulness and bronchial breathing may be heard over the affected base. In the parasitic cases, hydatids, actinomyces, and the Distomum pulmonale may be discovered in the sputum.

The hæmoptysis which supervenes upon bronchiectasis is easily recognised by the history of long-continued periodic expectoration of a characteristic odour and appearance. Even during the attack of hæmorrhage the numerous large fatty acid crystals may be found in bunches in the sputum, sometimes mixed with crystals of hæmatoidin.

Valvular disease, when it produces hæmoptysis, is easily recognised by the aid of the previous history and the presence of the cardiac murmurs ; a history of repeated attacks, extending over years, is often obtainable. Though the presence of valvular disease does not negative pulmonary tuberculosis, it should be remembered that this disease very rarely is found in

association with mitral stenosis, and hæmorrhage in mitral stenosis is often an early symptom.

Aortic Aneurism.—The possibility of this as a cause of hæmoptysis should never be lost sight of in doubtful cases. The bleeding may occur continuously for weeks in small amount when the sac of the aneurism weeps into the trachea. At other times it is intermittent, with irregular intervals ; but it may be even profuse and temporary recovery follow. When the sac ruptures into the trachea, bronchi, or pulmonary tissue, a rapidly fatal hæmorrhage follows.

Prognosis.—Beyond repetition of the statement that hæmoptysis is of itself rarely fatal, little need be said, as the prognosis is that of the disease which has caused the hæmorrhage in each case. In many instances it must be admitted that the bleeding, when moderate in amount, may do good by relieving congestive states of the pulmonary or bronchial tissue.

Treatment.—Absolute rest in bed is essential ; the best position is to lie upon the affected side with the shoulders elevated, when the source of the hæmorrhage can be located. Murray Leslie lays great stress upon the importance of turning the phthisical patient upon the side from which the bleeding comes, as by this means the blood is prevented from regurgitating into the bronchi of the sound lung, and asphyxia, which is the usual mode of death, is thus prevented.

Rest from mental excitement is of almost equal importance, and speaking should be forbidden in all severe cases. The room should be cool and well ventilated, and the bedclothing should be as light as the season will permit. The diet should consist of small quantities of cold liquid nourishment administered frequently, and ice may be permitted freely to relieve thirst and allay cough.

Drugs must be used with caution ; the routine administration of the host of so-called remote astringents, which were generally not only useless but in many ways injurious, is now being universally abandoned. Lead-salts, ergot, gallic and tannic acids, and the vegetable tinctures and infusions containing them, alum, digitalis, sulphuric acid, and iron preparations, may all be safely rejected. Morphia is of great value, but its use requires much discrimination, and it is perhaps true to say that it may hasten a fatal issue if given without due consideration of the dangers present.

When the hæmorrhage is continually being aggravated by efforts at coughing, in which small quantities of blood are brought up incessantly, a hypodermic injection, not exceeding $\frac{1}{4}$ gr., does great service. Where the amount of blood is copious, and where the purulent expectoration has been previously profuse from old phthisical cavities in weakened subjects, such a dose may be highly dangerous by stopping *necessary* coughing. It is in the hæmoptysis of the early

stages of phthisis that the best effects are obtained from morphia with the minimum of danger.

The main indication is to hasten and assist clotting at the ruptured orifice of the bleeding vessel, and this is often accomplished by nature during the syncope or fainting condition, when the circulation is at its very lowest ebb. The physician should aim at assisting nature, which he can do by reducing the blood-pressure in the pulmonary artery and by increasing the coagulable power of the blood.

The practitioners of last century accomplished the former result by opening a vein and bleeding till faintness occurred. The modern physician accomplishes the same result by a rapidly acting saline purgative, and in severe cases by the administration of a vaso-dilator like amyl nitrite, nitro-glycerin, or aconite.

Coagulation of the blood can be increased with certainty by the administration of calcium chloride, which is, moreover, harmless in ordinary doses. The best plan is to give 30 grs. immediately, dissolved in ½ oz. of water, and 5 grs. every quarter of an hour for eight doses. In this way, by giving a small and frequently repeated dose, the patient's mental anxiety is much more effectually relieved than if large doses were ordered at long intervals, many patients fearing that they may bleed to death before the interval has elapsed.

Adrenalin is highly recommended, and may be given in doses which do not raise the general blood-pressure but which are capable of arresting internal hæmorrhages of various kinds. Schlesinger, who found it valuable in purpura, typhoid ulceration, and even in hæmophilia, was unable to observe any beneficial effects from its use in hæmoptysis.

Other agents may be employed in conjunction with those already mentioned. The best of these is oil of turpentine inhaled in the form of vapour diffused through the air of the sick-room by pouring the liquid upon warm water contained in vessels about the patient's bed, or by sprinkling it on saw-dust. It never does harm, often allays cough, and seems to arrest effectually capillary oozing, which sometimes occurs from the relaxed mucous membrane of chronic bronchitis.

Ice to the chest is positively injurious if the application be continued for more than fifteen or twenty minutes at a time; occasionally it is very valuable when applied for this short period.

Ligatures temporarily applied to the limbs, with the view of retaining the blood in them and preventing its return to the heart, sometimes act like a mild venesection.

Saline injections or transfusion are indicated in desperate cases; artificial respiration may be tried where asphyxia threatens; and anæmia demands iron at a later stage.

HÆMORRHAGE, CEREBRAL.

(See under CEREBRAL HÆMORRHAGE, p. 173.)

HÆMOTHORAX

The extravasation of pure blood into the pleural cavity constitutes hæmothorax, and it should not be confounded with those numerous conditions in which a pleural effusion is stained with more or less blood.

Etiology.—Hæmothorax is usually the result of injury, and is therefore mainly of surgical interest ; it may occur during or immediately after the operation of paracentesis. Rupture of a thoracic aneurism, or the ulceration of a vessel in a malignant growth in the pleura, or the rupture of a thoracic vein owing to the pressure of a new-growth, are the usual sources of the blood, but it may be produced by a tubercular focus in the lung close to the surface, which has eventuated in the erosion of a pulmonary vessel, the blood passing into the pleural cavity instead of taking the ordinary route of an hæmoptysis. When blood escapes, owing to the necrotic changes which take place in pleural tuberculosis, it is nearly always mixed with pleural effusion.

The condition has been observed to follow the rupture of a pulmonary emphysematous patch situated on the surface of the lung, but in such cases, as well as in those caused by pulmonary tuberculosis, air generally also escapes, producing the condition known as *hæmopneumothorax.*

The diagnosis from ordinary effusion is difficult, and may be impossible without puncture. The prognosis will be that of the primary affection ; and the treatment that of ordinary effusion, unless clotting has occurred, when a free incision and drainage may be necessary. The following are the conditions in which pleural fluid may be found stained with blood—viz. in pleurisy associated with such diseases as chronic renal affections, cirrhosis of the liver, and malignant fevers, especially scarlatina, acute tuberculosis of the pleura, and notably in cancer, sarcoma, or lymphadenomatous growths of the pleura. Pleurisy occurring in purpura, scurvy, and hæmophilia is likely to result in blood-stained effusion. Sometimes the staining may be very considerable in an ordinary pleurisy, from rupture of the new vessels in the recently formed false membrane. All these conditions are grouped under the name *hæmorrhagic pleurisy*, which is used in contradistinction to that of hæmothorax.

HAY-FEVER

This affection, also known by the names of Summer or Autumnal Catarrh and Hay-asthma, manifests itself by symptoms of severe catarrh of the nasal or upper air-passages, usually accompanied by conjunctival irritation, and sometimes by attacks resembling, or identical with, bronchial asthma.

Etiology.—The disease affects chiefly the middle or higher classes, and is rare amongst the poor ; it may come on at any age, but most frequently before middle-life ; males suffer more frequently than females. A neurotic taint is often evident, and the susceptibility seems sometimes to run in certain families. It is most frequently met with in dwellers in cities and in those engaged in mental pursuits. Nasal disease as chronic hypertrophic rhinitis, polypi, and malformations as deflection of the septum, certainly appear to be strong etiological factors, since the disease has been known to be cured by removal of these conditions.

The main exciting cause is undoubtedly the irritation produced by the pollen of grasses ; recent researches in the domain of therapeutics have demonstrated this fact in a striking manner. Dust and pollen, and irritants from many other sources, smoke, &c., produce the symptoms ; but the most interesting fact recently discovered in association with the etiology of hay-fever is that the attacks are not caused by the mechanical irritation of the grass pollen-grains, since those which excite the symptoms have, according to some observers, a perfectly smooth surface.

Dunbar has isolated a toxic proteid substance from the pollen of certain grasses which, when applied to the eye and nasal membranes, quickly produced the characteristic symptoms of the affection in persons subject to hay-fever, whilst in persons who had not suffered previously, no effects were produced. Semon, who has corroborated the Hamburg professor's experiment, regards the toxin as undoubtedly specific.

Dunbar, by injecting the toxin into the circulation of animals, chiefly thoroughbred horses, has obtained a serum or antitoxin which, when applied to the eyes and nose, immediately arrests the local symptoms produced by the application of the toxin. Semon, Lübbert and Prausnitz, Mayer, McBride, Somers, and others have established the efficacy of the serum in the treatment of several hundreds of cases of hay-fever, the vast majority of which were speedily relieved by dropping the liquid serum into the eye, and applying, as a snuff, the powdered preparation to the nasal surface. It is interesting to note that Somers prepared his serum from the blood of animals injected with the pollen toxin of golden rod.

Pathology.—It may be taken as practically proven from the above researches, and from the facts previously mentioned, that there are at least three factors present in hay-fever: (1) A neurosis which may be ultimately found to be identical with that present in ordinary bronchial asthma; (2) a hypersensitive or irritable condition of the nasal mucous membrane; and (3) the presence of an irritant, the common source of which is the proteid poison existing in the pollen of various grasses. The complete removal of any one of these factors, as pointed out by Lockard, will cure or prevent the disease.

Symptoms.—These set in suddenly, during the season of the year when pollen is abundantly present, with severe coryza, redness of the eyes, sneezing, and sometimes irritating cough. In those patients where the neurotic factor is obviously in evidence, Wallis points out that the psychical element shows itself by the periodicity of the affection and its regular occurrence when expected or dreaded, and its absence when the thoughts are otherwise fully occupied. According to Thost, about two-thirds of the patients exhibit true asthmatic symptoms. Often there is impairment of taste, smell, and hearing, and occasionally some fever is observable, and usually much depression of spirits and an acute sense of great discomfort. The symptoms vary much in severity, being aggravated by exposure and local environment during the attack, which may last for over a month.

Treatment.—Residence in a place devoid of vegetation at the summer or autumn season is both prophylactic and curative; but such spots are difficult to find, except upon mountain-tops and on the open ocean. Occasionally seaside resorts are beneficial.

The usual remedial agents indicated in asthma are beneficial, and attention should be paid to the condition of the nasal mucosa. Local applications are legion; carbolic acid, quinine, cocaine, adrenalin, &c., give but fleeting relief, and often afterwards cause aggravation of the symptoms. Marked improvement often follows the cauterisation of the septum as practised by Francis, of Brisbane, and the writer has seen several cases treated successfully by Dr. Joseph Nelson, who employs this method.

At present, the best of all remedies seems to be the new serum of Dunbar, repeatedly used locally as a prophylactic before the advent of the pollen-season, and before each exposure in the open country, and as soon as the first symptoms show themselves. The serum should never be employed hypodermically.

HEADACHE

This must be regarded merely as a symptom of a large number of diverse diseased conditions or morbid states. Megrim is described under its own heading ; neuralgic pain occurring in the scalp in areas corresponding to the distribution of nerve-trunks should not be included under headache.

The perception of pain, according to Gowers, is related to the activity of certain nerve-cells in the cortex of the brain, and not to consciousness of pain located in the lower centres. This is evident in the case of pains originating in the limbs, for example, and he reasons that meningeal pain is perceived by the activity of the cortical cells which receive impressions from the irritated meningeal nerves. Probably the brain-tissue itself has sensory representation in the cortex. Headaches may be classified in two great divisions—*structural* or *organic*, and *functional*. In the first group are included those cases dependent upon diseases within the cranium, as meningeal inflammations of various kinds, traumatic, syphilitic, tubercular, &c., the growth of tumours, abscesses, hæmorrhages, inflammation of the cerebral tissue, as encephalitis and disease of the vessels and bones. The characteristics of the pain arising from structural disease vary ; but, as a rule, the pain is more or less constant and usually very severe, and possesses a feature which is almost diagnostic : it usually prevents sleep. Though its localisation is as variable as its cause, its tendency is to remain confined to a particular region ; and this must not be accepted as corresponding to the actual seat of mischief, as, for example, it is a recognised fact that cerebellar disease may cause constant frontal headache.

The pain, like that in all severe headaches, is increased by temporary changes in the blood-pressure caused by coughing, stooping, or muscular exertion ; but, though liable to severe exacerbations, it seldom entirely disappears in the intervals. Vomiting and optic neuritis are often found associated with it, though these symptoms are occasionally to be met with in the severe headache caused by renal disease and some anæmic conditions. Local deep tenderness on percussion of the scalp is often present, and is characteristic.

Functional headaches are caused by extracranial conditions so numerous and varied that classification becomes difficult.

1. *Toxæmic* headache constitutes the great bulk of all the cases met with in practice. It is found in its most typical acute form in the various fevers, notably in small-pox, typhus, and influenza. In a small percentage of typhoid-fever cases in the early stage the cephalalgia may be almost maddening in its extreme severity. All inflammatory conditions are

associated with more or less of this toxæmic headache, though occasionally one meets with a patient who affirms that he has never known what headache means.

Toxæmic headache is generally frontal, and rarely unilateral ; it is a deep-seated diffused pain, often dull, but never quite continuous save in the very severe form met with in Bright's disease, when it presages a uræmic convulsion.

Diseases of the liver, obstinate constipation, gout, diabetes, dyspepsia, especially if intestinal, and all conditions tending to produce auto-intoxication, cause toxæmic headache. Poisons introduced from without, as lead, foul or re-breathed air, alcohol, ethers, nitrites, quinine in large doses, opium, and many other drugs, act in a similar manner.

2. *Congestive* headache is met with in plethoric states, suppressed menstruation, and in conditions when the general blood-pressure is high, as in arterio-sclerosis and in cases of great mental excitement. In these instances it is usually associated with flushing of the face, contracted pupils, and marked throbbing. In passive congestions, produced by valvular heart-disease, and the pressure of tumours or other constrictions outside the skull which interfere with the return of the venous blood from the brain, the pain is generally of a dull constant character, which, however, like every form of severe headache, is aggravated by stooping and cough. This latter may, when incessant, be the cause of very severe cephalalgia and giddiness. To this class probably belongs the headache following epileptic seizures.

3. *Anæmic* headache is a constant feature in chlorosis, and in anæmia from whatever cause ; the pain is usually dull and diffused, but it may be confined to the frontal region or the vertex, or may be post-orbital, as in megrim. The pupils are generally dilated ; vertigo or dizziness, tinnitus, and other symptoms of anæmia are often present. To this type belongs the severe headache met with from brain-fag following prolonged mental exertion and anxiety, and debilitating causes, as hyperlactation. Anæmic head-pain is usually relieved on assuming the recumbent posture.

4. *Hysterical* or *neurasthenic* headache is an ill-defined type ; it is superficial in character as a rule, and located in the frontal or occipital region, and is generally more or less diffused and continuous. With the pain are usually associated numerous sensations, which the patient often does not differentiate from real pain, as a sense of fulness, pressure, bands, &c. Sometimes it is found impossible to get from the patient information which enables the physician to decide whether the pain is in the scalp or inside the cranium. Gowers gives the name of *neuralgic* headache to the common form in which the pain seems to be superficial in part or altogether, and limited to an area which does not correspond to the distribution of a

superficial nerve. This is very different from the severe circumscribed painful sensation as if a nail were being driven through the skull (*clavus hystericus*). Purves Stewart points out a valuable diagnostic feature in neurasthenic headache—it is worse when the patient is tired, and it improves on resting.

5. *Pericranial* headache possesses features closely resembling those so commonly seen in the neuralgic headache of hysterical patients; thus *rheumatism* affecting the nerve-sheaths as they pass through the cranial apertures, or involving the pericranium or the occipito-frontalis and its aponeuroses, may produce a headache which is associated with scalp-tenderness on pressure, and is aggravated by movements of the eyebrows. This so-called *rheumatic* headache is identical in most of its characters with that caused by gout and syphilis.

6. *Reflex* headache. This constitutes a very important clinical group which has lately received much attention. To it belongs the so-called *ocular* headaches, which are the result of eye-strain produced by errors of refraction, weakening of the ciliary muscle, and insufficiency of the globe rotators preventing normal fixation.

Snell points out that so-called '*academy* headache' is produced by the unaccustomed strain upon the elevators of the eyes, caused by prolonged gazing at pictures above the line without throwing back the head; bicyclists suffer from a similar head-pain by riding with the head low down; and miners are affected in the same way.

The majority of the cases of ocular headaches are produced by hypermetropic astigmatism; others are the result of myopia, iritis, and glaucoma. The headache is usually occipital in muscular derangements causing strain in fixation in order to prevent double vision; whilst it is generally supra-orbital in astigmatism.

There is also a *nasal* group in which naso-pharyngeal adenoids cause reflex irritation in mouth-breathers; but any disease of the turbinated bones or of the sinuses in the frontal, ethmoid, sphenoid, or maxillary bones may cause 'nasal' headache, which disappears on removal of the cause.

Caries of the teeth and ear-troubles often cause headache in which the site of the pain may be varied.

7. *Gastric* headache, so called, is usually occipital. It may be the result of reflex irritation; but in some cases it may belong to the toxæmic group, owing to auto-intoxication by products formed during perverted digestion, or both varieties of causation may be acting together. This is also seen in Bright's disease, where there may be a hyperæmic condition of the diseased cerebral vessels, as well as a toxic state of the blood the result of diminished elimination. This dual nature of the causation must never be lost sight of in the investigation of obscure cases.

Of even more importance is the underlying neurotic state or individual peculiarity ; it is a well-recognised fact that one patient will suffer no inconvenience from an error in refraction which in another would cause intense cephalalgia. This is also seen in the reflex headache sometimes caused by uterine displacements and ovarian irritation.

Diagnosis.—The chief points in the diagnosis have been included in the above description ; every case must be carefully investigated, and of greatest importance is the recognition of the structural type depending upon serious intracranial organic disease. As already mentioned, the severity and constancy of a headache, associated with vomiting and optic neuritis, are almost pathognomonic of grave mischief within the cranium. The fact next in importance is the condition of the optic apparatus, and perhaps that of least importance is the site of the headache itself. The urine should be invariably examined. In children the possibility of tubercular meningitis must be always remembered. The diagnosis of headache from *megrim* will usually be easily made upon contrasting it with the other characteristic symptoms of this disease.

Prognosis.—This will be the prognosis of the primary condition. When the cause is removable, the forecast is always good ; but when the opposite condition exists, the most that can be hoped for is some degree of alleviation of the cephalalgia.

Treatment.—This should never be upon empiric lines : drugs or other remedial agents should not be prescribed till the cause has carefully been sought out. This, on being found, will suggest a rational basis for treatment. Thus ocular headache yields at once to correction of errors in refraction ; anæmia, though more slowly, yields to iron ; liver and intestinal troubles speedily disappear under the influence of mercurials and saline purgation.

Congestive headaches also yield to smart purgatives, and the principle upon which toxæmic head-pain should be treated is obvious : eliminatory measures—cutaneous, renal, and intestinal—should be pushed, as described under Bright's disease, though in this latter condition diuretics are not available.

The toxæmic cephalalgia of fevers, as a rule, cannot be treated by heroic purging and sweating, though diuretics may be freely employed. In these cases one must be content to wait till the fever subsides, and usually the severity of the pain fortunately diminishes as the disease progresses ; in the meantime relief may be obtained by the ice-cap and by smart counter-irritation to the nucha—measures of great value as palliatives in meningeal and congestive headaches.

Drugs which possess distinct analgesic powers are now numerous ; foremost amongst these are the coal-tar derivatives—antipyrin, antifebrin, phenacetin, exalgin, &c., and

salicylates, aspirin, caffeine, bromides, &c. These may be pressed into the service for the relief of head-pain in the neurasthenic and neuralgic types, and in those produced by brain-fag, nervous debility, and the various anæmias, during the period of treatment directed towards the improvement or removal of the primary condition. Under 'Megrim' will be found mentioned other agents suitable for the palliation of most of the types of headache already mentioned ; the treatment of each primary condition is referred to under its own heading.

Little need be said of narcotics : morphia and opium are as a rule harmful, save for producing sleep in the headache of fevers, when this causes insomnia. Bromides are generally useful in combination with the coal-tar analgesics, and this is the usual routine treatment for the relief of pain in the meningeal and intracranial forms where morphia is especially harmful. Large doses of iodides often afford considerable relief in headaches caused by intracranial syphilis.

HEART, ANEURISM OF

This is a rare condition, chiefly of pathological interest only, since few, if any, of the cases are capable of being diagnosed during life.

Pathology.—The sac of the aneurism consists of a pouch or sacculus, lined by an extension of the endocardial membrane, situated in the great majority of cases in the wall of the left ventricle near to its apex ; occasionally the site is the ventricular septum. The walls are made up of the endocardium, more or less fibrous tissue mixed with cardiac muscle, and, in long-standing cases, of the pericardium which may be dense and greatly thickened. In these latter cases the muscular tissue may have entirely disappeared, except at the base of the tumour.

The pouch is in open communication with the interior of the ventricle, but in some instances there may be a true sacculated dilatation communicating with the ventricle by a narrow mouth. The tumour varies in size from that of a small marble to that of the closed hand or fist, and in its interior are found laminated fibrinous layers and recent blood-clots.

Etiology.—Any agent which causes weakness or injury to a localised area of the cardiac wall may become the starting-point of a bulging which gradually yields to the blood-pressure, till finally the pericardium is reached and the tumour appears, on the outside of the heart.

Chronic inflammatory changes, as myocarditis, associated with fibrous degeneration, often the result of coronary disease, are the most common causes of the original weakness in the ventricular wall ; but the condition is sometimes the result of

an acute endocarditis of septic origin, and it may be preceded by the formation of an abscess. Fatty degeneration and the changes induced by syphilis are supposed to be the causes in some cases, but fibroid degeneration accounts for the great majority of all the carefully recorded instances of this rare condition.

Symptoms.—The symptoms are variable and in no way distinctive. Murmurs were present in a considerable proportion of the cases where cardiac aneurisms had been found after death. Dyspnœa, heart-pain, and palpitation have been noticed, and in some cases the tumour has caused bulging in the apical region, which has been mistaken for dilatation and hypertrophy. The course of the disease varies: sometimes death occurs suddenly by rupture into the pericardium, in other cases the dilatation has proved fatal by its mechanical effect upon the circulation. In rare museum-specimens the sac is seen to be spontaneously obliterated or calcified.

Aneurisms sometimes form on the valves of the heart in ulcerative endocarditis. They are generally found upon the ventricular aspect of the semilunar valves; the weakened or eroded spot bulges under the pressure of the blood in the aorta, which causes the tumour to project into the left ventricle. When these aneurisms rupture, if death does not occur at the time the perforation causes extensive injury to the valve-substance, which may ultimately destroy its function.

Aneurism of the coronary arteries is a rare condition, usually the result of atheromatous changes, and, like cardiac aneurism, does not cause any symptoms by which it can be recognised during life.

HEART, ATROPHY OF

This condition is of little clinical interest compared with its opposite—hypertrophy. The heart is found atrophied in most chronic wasting diseases, and after death its weight may be reduced to half the normal average, the withered organ being found to weigh as little as four ounces. Atrophy of the left ventricle may follow simple stenosis of the mitral valve. The condition to which the term 'atrophy of the heart' is most correctly applied is that in which the peculiar form of change in the myocardium which is known as *pigmentary degeneration* or *brown atrophy* has occurred. This change is present in the hearts of many elderly subjects, and must be regarded as a senile manifestation; but it may be also found in cachectic or wasting conditions. The colour of the atrophied organ is a dark brownish red, and it is tougher than normal, smaller in size, and weighs less. The pigmentation is due to reddish-yellow hæmatoidin granules around the nuclei, in the interior of the muscular fibres. These latter are reduced in number

and in size. The condition is one which leads to no specific symptoms, save perhaps an enfeebled circulation ; but its presence may be assumed in a patient exhibiting marked senility, with a weak pulse, diminished area of cardiac dulness, and a circumscribed feeble apex-beat. It must be remembered that, owing to disease of the arteries of the body, the hearts of aged subjects are often considerably enlarged.

Though fatty degeneration of the heart is associated with wasting of the muscular fibres, a general atrophic result is not witnessed, save in comparatively rare examples only met with in aged subjects. The explanation is found in the fact that there is always more or less new fibroid-tissue growth accompanying the degenerative change in the muscular fibres. The atrophic changes described as *fibrillary* in which the longitudinal striation of the cardiac muscular fibre is greatly accentuated, and the so-called *fragmentary atrophy* or *segmentation* in which the fibres separate at the cement-line or break across, are not of any practical clinical interest, and by many are supposed to be the result of *post-mortem* change.

HEART, DEGENERATIONS OF

These affect the myocardium in various ways. The most important are: (1) The *Fatty* ; (2) the *Fibroid* ; (3) the *Brown Pigmentary* degeneration dealt with in the preceding article ; (4) the *Cloudy Swelling* or *Parenchymatous* degeneration—*i.e.* the change observed in the protoplasm of the cells and fibres caused by the poisons of infectious diseases, diphtheria, Bright's disease, &c. ; (5) the *Hyaline* degeneration (of little clinical interest save that it is sometimes found in association with toxæmic conditions).

FATTY DEGENERATION

By this is meant the change in the muscular fibres, whereby their protoplasm is converted into fat-elements.

Morbid Anatomy.—The degeneration first appears in a granular form in the fibrils, owing to the deposition of minute granules of fat ; at a later stage the nucleus is also broken up.

The degenerative change may affect the entire myocardium ; but often it occurs in lines or patches, in the form constantly seen by the naked eye in malignant anæmia: this constitutes the so-called 'tabby-cat' striation, or 'thrush's breast' mottling. When the distribution is general the organ is softened and flabby and usually distinctly enlarged, though its weight may be but slightly increased ; the cavities are somewhat dilated, and the organ is a light-brown or buff colour, and easily lacerated by the fingers. The endocardium and the pericardium usually are unaffected, but the papillary

muscles show marked change, especially in the left ventricle, where their striped appearance has been described as the 'tiger heart.'

Etiology and Pathology.—The causes which produce this condition are numerous, and may be either *local* or *constitutional.* Amongst the local causes, by far the most important is disease of the coronary arteries. This, by interfering with the blood-supply of the cardiac muscle, induces fatty and fibroid changes; hence both forms of degeneration are usually found together, but in the senile heart the fibrous degeneration may be altogether absent.

Another important etiological distinction is usually observable when the change is the result of coronary disease: the fatty degeneration is usually confined to certain areas, and the softening is rarely uniformly distributed. The usual causes of the coronary disease are syphilis, gout, alcoholism, and strain, which tend to produce atheroma; finally, thrombosis and embolism may supervene, and death result. This is often the cause of a suddenly fatal issue, since the coronary vessels have but few anastomoses, and may be regarded as end arteries; blocking of one of them is sufficient, therefore, to produce the so-called 'anæmic necrosis,' or white infarct, which sometimes occurs in fatty degeneration of the heart.

Another local cause is the myocarditis which is often met with in old valvular lesions when dilatation supervenes in the stage of failing compensation.

The constitutional causes of fatty degeneration of the heart are varied and numerous; thus anæmias of all kinds, especially the pernicious types, induce it, and more or less temporary fatty change is constant in chlorosis. It is found in all wasting diseases and cachectic conditions like cancer, tuberculosis, &c., and it is a part of the senile condition. All infectious fevers, especially typhus, are associated with grades of fatty degeneration, which, however, are rarely permanent. The acute toxæmic condition, which is part of the diphtheritic process, is the most grave of these, and often causes an acute degeneration of the muscular fibres which ends in a fatal syncope. Poisons like phosphorus, arsenic, antimony, lead, and alcohol, and probably also the toxic products of such diseased actions as gout and nephritis, may induce the condition; of these, phosphorus is by far the most powerful.

The disease affects males more than twice as frequently as it does females, and the usual period of life at which it first makes its appearance is after fifty.

Symptoms. — These are sometimes entirely latent till dilatation occurs, but usually the increase in the capacity of the cavities is gradual, and keeps pace with the degenerative change. There is generally some enlargement of the heart, and, as already seen, the muscular fibres are to a smaller or

greater degree converted into fat. These two facts give the key to the signs and symptoms. There is a small and enfeebled pulse, which is markedly irregular, a somewhat diffused and feeble apex-beat, which is displaced outwards, and the area of cardiac dulness is appreciably enlarged. Upon auscultation the first sound is muffled or indistinct and approaches in character the qualities of the second ; there may be irregularity either in the length of the pauses, the intensity of the sounds, or the strengths of the ventricular contractions when several cardiac revolutions are contrasted with each other. Exertion causes a considerable amount of dyspnœa, and there is *slight* lividity of the ears and blueness of the lips. With these disturbances of the respiratory and cardiac rhythm Cheyne-Stokes breathing may become evident, but usually it does not occur till the late stage of the disease : the respirations become rapid, and more and more shallow, till they ultimately stop for perhaps half a minute ; then slowly and almost imperceptibly they start again, becoming quicker and fuller for a short period, to pass once more into shallow efforts, which again end in a dead pause, after which the phenomenon recommences. Usually this type of breathing occurs during sleep, and the patient often starts up awakened by the pause ; it is not peculiar to fatty heart, but is met with in uræmia and cerebral conditions, and in aortic disease.

Anginal attacks are common and, like Cheyne-Stokes breathing, they are of grave omen, indicating serious coronary disease. Syncopal seizures are also frequent, and the unconsciousness may be profound and protracted; so that the attack resembles an apoplectic fit of short duration, in which stertor, coma, and convulsive twitchings may be observed. In some cases there is marked infrequency of the pulse, which the writer once saw fall to fifteen. As a rule, in the attacks pallor is much more common than lividity, and cyanosis is very rarely seen unless considerable dilatation has supervened. A certain amount of dyspnœa and a choking sensation or suffocative feeling, associated with giddiness, are nearly always present in marked cases when rising ground is being traversed. Upon these occasions the pulse becomes very rapid and the irregularity increases, sighing respiration often following after the cessation of the dyspnœa.

Cardiac pain is not usually a prominent symptom, unless during the anginal seizures. A slight amount of œdema of the ankles is often present, but extensive dropsy rarely occurs. though a murmur indicative of mitral regurgitation is observed in many cases ; albuminuria is unusual. Dyspepsia, irritability of temper, or even mental symptoms of a marked type, may occasionally appear, and more or less inability to pursue any difficult train of thought, except for a brief period, is usually present. The duration of the disease is very variable.

Some patients fortunately situated in an environment free from care and pressure of every sort may live for very many years; in other cases the progress of the malady from the first appearance of serious symptoms to the fatal termination may occupy but a few weeks or months. There are, however, good reasons for believing that in such cases the degeneration had been long latent, and the results of *post-mortem* examinations prove this. Moreover, some rare cases are entirely latent till a sudden fatal issue supervenes, and not unfrequently advanced fatty degeneration of the heart-muscle has been found after death from other causes, when its existence had never led to any symptom during life.

Death in the majority of cases is sudden, either from syncope, coma, angina, or rupture of the heart; in rare cases the patient may slowly sink from a progressive asthenia.

Diagnosis.—From what has been said of the latency of the condition, the diagnosis might seem to be inscrutable in many cases, but this really is not so: such cases probably might have been discerned by a careful physical examination, though the patient was unconscious of any cardiac weakness. Every physician of experience can probably recall instances where he has been morally certain from the discovery of physical signs that the disease was present in a patient who has consulted him for some ailment totally unconnected with the disease. The presence of arcus senilis is of little or no diagnostic value.

The greatest difficulty arises in the differentiation of *fibroid degeneration* of the heart, and the diagnostic points will be discussed under the heading of that affection. *Angina pectoris* is recognised by the agonising attacks of pain which constitute the chief, and sometimes the only, feature in the clinical picture, the patient being often free from all signs of disease save the arterio-sclerosis, which is perhaps always present, but which is not always capable of demonstration. The condition of the angina patient between the attacks, with his normal or high-tension, strong, and regular pulse, affords a marked contrast to that of the subject of fatty degeneration, with his irregular, small, and feeble pulse, muffled heart-sounds, and signs of cardiac dilatation. The history of syncopal attacks, or frequent faintness associated with coma, is not found in angina.

From *valvular lesions* the diagnosis is usually easy. The presence of the characteristic murmurs, associated with a much greater degree of hypertrophy and, if compensation has already failed, the presence of dilatation of a much greater extent than is ever seen in the fatty heart, are characteristic. Moreover, the prevailing symptoms are those of venous stagnation, seldom met with in cases of fatty heart. It must, however, be remembered that this latter condition may

supervene upon the hypertrophy of valvular disease, in which case the history, combined with a study of the symptoms and a recognition of the failure of any response to the treatment by cardiac tonics, will clear up the diagnosis.

Fatty infiltration must not be confounded with fatty degeneration, and a brief description of this condition may be here recorded. It consists in a deposition of true adipose tissue over the surface of the heart, starting in the sub-pericardial fat, of which it is a simple overgrowth. It is deposited between the muscular fasciculi, chiefly following in its distribution the arterial trunks, and in well-marked cases it extends between the bundles of muscular fibres from the pericardial to the endocardial membrane. The entire organ may thus become buried in a thick layer of fat ; usually the right ventricle is more generously enveloped than the left. Unless in advanced cases, the muscular fibres may be seen in their normal condition amidst abundant fat-cells, but at a later stage the fat by its mechanical pressure causes atrophy of the fibres. Occasionally the compressed and atrophied muscle-fibre may become the seat of true fatty degeneration. In other cases, fatty infiltration may be a secondary change following fatty degeneration in anæmia or some wasting disease. This condition of fatty infiltration, fatty overgrowth, or cardiac lipomatosis, as it is also called, is generally a part of obesity. It is found in middle-aged large eaters, who take little open-air exercise and a fair amount of alcohol, and it is prone to occur in subjects of chronic emphysema and other lung-conditions in which the blood is poorly oxygenated.

There is always more or less dilatation of the chambers of the flabby enlarged organ, which gives signs of increased cardiac dulness, with a feeble and diffused apex-beat which may be indistinctly felt towards the xiphoid. The sounds are muffled and weak, and when marked dilatation has supervened there is usually a soft apical systolic murmur.

The symptoms are those of enfeebled circulation in stout flabby persons. Shortness of wind on slight exertion, associated with a marked tendency to sweating, and a quick, weak, but regular pulse of low tension, are present. The condition is much more common in men than in women, and is rare before fifty. Other symptoms, such as those noticed in true fatty degeneration, may be present, and since both diseases may occur together the differential diagnosis is often a matter of difficulty or conjecture. The chief characteristic feature is the presence of marked general obesity.

Death may occur suddenly from syncope or rupture, though this is much less liable to happen than in fatty degeneration ; and probably when the affection ends fatally always some degenerative change is also present.

The treatment of fatty infiltration of the heart is that of the general obesity always present.

Prognosis.—In fatty degeneration the prognosis will depend upon the cause of the disease, but the probability of sudden death is never to be forgotten. In the acute cases following anæmic states and fevers and the action of mineral poisons the outlook is usually good, but the diphtheritic cases are very treacherous. When the condition is due to organic disease of the coronary vessels, constituting the clinical picture already described, the ultimate prognosis is only a question of time, but by judicious care the fatal issue may often be postponed for many years. In simple fatty infiltration the prognosis is usually very good when the patient can be depended upon to carry out steadily the treatment for obesity.

Treatment.—In the acute toxæmic and anæmic cases the degeneration disappears upon the removal of the cause, hastened by rest, good feeding, and iron. In the diphtheritic form, strychnine hypodermically must be resorted to early. In the chronic type produced by coronary disease, palliation of the main symptoms and some prolongation of life are all that can be hoped for. Everything in the environment of the patient which puts strain upon the degenerate organ must be strictly avoided ; his life must be made as free from worry or pressure as his circumstances will permit. Regular hours, and habits of periodical resting in the daytime, must be instituted. As much of his time as is possible should be spent in the open air ; all exercises must be gentle and methodical, and interrupted by brief periods of enforced rest. Rising ground and stairs must be avoided or negotiated with great slowness and deliberation, climbing being entirely out of the question.

The diet should be rich in nitrogenous material, and four or five small meals spread evenly over the day must take the place of the late heavy dinner, or still more objectionable hearty supper. Starches and sugar should be used sparingly, and alcohol only taken in very small quantities in the form of light wine. Tobacco, if used at all, must be indulged in most sparingly.

Of drugs, the best, and indeed the only one of real value, is strychnine ; in the presence of syncope it should be given hypodermically. Two drugs are recommended by several authorities—*i.e.* phosphorus and arsenic ; both of these agents are regarded as capable of causing the disease, hence it is very doubtful if either should ever be employed.

Cardiac tonics, digitalis especially, are very frequently administered as routine remedies in every case of cardiac failure ; it is highly probable that they often hasten death by causing rupture of the ventricle.

The constriction which these agents produce in the peripheral vessels causes a rise of blood-pressure, against which the degenerate cardiac muscle must put forth increased power in order to overcome the resistance in front, in which case the rapidly distended ventricle may burst. This danger is only theoretically lessened by combining vaso-dilators with the cardiac tonics, since the effects of the former are but transient, whilst the rise in the peripheral resistance is practically continuous. Strychnine possesses only slight influence over the peripheral vessels, unless when given in very large doses; though Cushny attributes its beneficial action in heart-weakness to its power of raising the blood-pressure in this manner.

Anginal seizures are relieved by amyl and other nitrites, and syncopal attacks by ammonia, alcohol, and other cardiac stimulants when strychnine is not available. Iron is always useful when given in combination with strychnine. Oxygen is recommended highly by Sir Douglas Powell when the Cheyne-Stokes respiration is present.

The treatment of fatty infiltration of the heart is the treatment of obesity, but when signs of degeneration appear, exercises of all kinds must be seriously curtailed ; massage is valuable when these must be prohibited. Nauheim treatment is only available with safety in the earlier stages of either condition.

FIBROID DEGENERATION OF THE HEART

This condition is known by several names, the mention of which demonstrates the difficulties existing in regard to its pathology ; thus it is also known as *fibrous myocarditis, chronic interstitial myocarditis*, and *fibroid infiltration*. By whatever name it is called, or upon whatever theory its etiology and pathogeny are to be explained, it is recognisable as a distinct affection, which can often be ascertained during life.

Etiology.—By far the most frequent and important cause of this form of myocardial affection is disease of the coronary arteries of the obliterative type. Its etiology therefore is practically identical with that of fatty degeneration of the organ, and, as might be expected, both conditions often exist together.

Pulmonary lesions of an obstructive type, as emphysema and fibroid phthisis, old valvular affections, and pericarditis, especially with adherent pericardium, by their direct or indirect effect upon the nutrition of the heart-muscle may induce this condition. It is always, therefore, present in old-standing cases of hypertrophy from whatever cause, when compensation has failed in its chronic struggle to keep the mechanical balance effective. It is probably also sometimes caused by alcoholism, gout, and syphilis.

Like fatty degeneration, it may be either general or local in its distribution in the myocardium. The cause of localised areas of fibroid degeneration is a blocking by embolism, thrombosis, or calcification of one of the coronary branches ; indeed, when one of these end arteries becomes blocked, degenerate myocardial changes must occur, unless the vessels of Thebesius, which open directly from off the cavities of the heart, be sufficient to carry on a vicarious circulation sufficient to nourish the muscular fibre. It is the existence of these vessels which often prevents local death or anæmic necrosis of a portion of the ventricular wall.

The affection, like fatty degeneration, is much more common in males, and occurs after fifty.

Pathology and Morbid Anatomy. — The characteristic feature of the change is an overgrowth of the interstitial fibrous tissue of the myocardium, the degeneration of the muscular fibres being secondary to this. The question of whether the hyperplasia or fibrosis is of the nature of a true degeneration, or of a low form of inflammatory action, is one only of theoretical interest. Those who hold the former view call the lesion by the name at the heading of this article, whilst those who regard it as of inflammatory origin call the condition 'chronic myocarditis.' Powell holds a slightly different view, and regards the affection as a fibroid *infiltration* closely analogous to fatty infiltration. There is still another theory, which accepts the muscular atrophy as the primary lesion, the overgrowth of fibrous tissue being regarded as a compensatory or secondary result. Whichever view be accepted, confusion will be prevented by regarding, in the present state of our knowledge, the condition under consideration as practically identical with what is sometimes separately described as chronic myocarditis.

The overgrowth of fibrous material leads to an increase in the bulk and weight of the heart ; and though the walls of the ventricles are really strengthened by the new tissue, owing to its lack of vital elasticity it yields slowly to the pressure of the blood in systole, and the chambers thus dilate ultimately.

In long-standing cases where the fibrosis is extreme, as in the apex of the left ventricle in coronary disease, the muscular fibres may have entirely disappeared, their place being occupied by dense scar-like tissue.

In a small percentage of cases the interstitial myocarditis may be acute ; and when pus appears, this is caused by micro-organisms which have produced an endocarditis of the ulcerative or pyæmic type. Acute interstitial myocarditis not ending in pus is a part of the rheumatic process, which extends from the endo- or pericardium, and it may be met with in the infectious fevers and diphtheria. Such causes are only of

practical interest when they lay the foundation of areas of fibroid degeneration appearing at a later stage, and which may be the cause of cardiac aneurism, as already described.

Symptoms.—The circulatory troubles caused by chronic myocarditis or fibroid degeneration (since, as just explained, these terms may be taken as synonymous) closely resemble those already described under 'Fatty Degeneration.' There is evidence of feebleness of the ventricular contractions, as shown by a small and usually low-tension regular pulse, which contrasts with the comparatively strong and diffused apex-beat of the enlarged organ. When marked dilatation has set in, the pulse may be very irregular; breathlessness, slight œdema of the ankles, anginal seizures, or attacks of cardiac asthma are common. Death may be sudden, as in fatty heart; this is usually the result of coronary plugging. Occasionally a severe attack of breathlessness, pallor, or lividity, with pain and grave symptoms of impending death, may suddenly supervene in fibroid heart, and the patient recover and for a considerable period enjoy a fair amount of health. In these cases probably some coronary branch has been occluded, and the vicarious circulation, by the aid of the vessels of Thebesius, may have been sufficient to keep the degenerated cardiac muscle alive. In some cases an advanced degree of fibroid change has been found on *post-mortem* examination in subjects who had never suffered from any cardiac disturbance.

Diagnosis.—From the failing compensation of *valvular* disease this is not easy, unless the history is clear, but as a rule the hypertrophy and supervening dilatation of valvular disease are much greater than in myocardial degeneration; and in this latter, evidence of fibroid change in the systemic vessels will be usually present.

It must always be remembered that the dilatation supervening upon all conditions of weakness of the cardiac muscle may give rise to a systolic mitral murmur; but the presence of the *bruit de galop*, which is one of the most valuable signs of failing muscle-power, may exist without any murmur. Its presence under such conditions in the so-called false hypertrophy of fibroid degeneration of the heart is of great diagnostic value in the elimination of valvular disease.

Fatty degeneration is eliminated with great difficulty; often it is associated with the fibroid form under consideration, when the symptoms of each are intricately woven together. Speaking broadly, the following differential points may probably be of some value in discriminating between typical advanced examples of each condition. The degree of enlargement is usually greater in the purely fibroid heart, the pulse is less likely to be so irregular and usually does not fall so low in frequency, and thickening of the radials is perhaps more

likely to be noticed than in fatty cases. Syncopal attacks, with coma and muscular twitchings, Cheyne-Stokes breathing, and constant drowsiness, are more characteristic of the fatty heart. Generally speaking, the dilatation of fatty disease ends fatally before the occurrence of the marked lividity, œdema of the legs, and signs of mechanical interference with the circulation which are often seen in the fibroid form. Many authorities maintain that it is not possible to differentiate between the two diseases.

Prognosis and treatment are practically the same for each affection, and they are equally uncertain and unsatisfactory.

Infiltration of the myocardium, pericardium, lining membrane or valves, with deposits of lime-salts, is not uncommon, but it is of little clinical importance. The disease of the coronary vessels producing the fatty and fibroid degeneration just described is usually associated with this form of calcification, which was formerly called calcareous degeneration.

HEART, DILATATION OF

In this condition one or more of the cavities of the heart are found to be increased in capacity—*i.e.* the dilated chambers, irrespective of the thickness of their walls, are capable of containing a larger quantity of liquid than they should hold in the normal state. Rarely are the walls of the average standard of thickness; most frequently this is increased; sometimes, however, it is diminished. The condition is therefore most frequent in which the walls are hypertrophied and the chambers dilated; this is called hypertrophy with dilatation, or eccentric hypertrophy of the heart. For this reason the subject of dilatation cannot well be *pathologically* considered as distinct from hypertrophy.

Clinically, however, the case is different. Speaking broadly or generally, the physician usually only associates in his mind the state known as cardiac dilatation with those cases where, owing to failure of muscular power, the dilated ventricle becomes unable to empty itself, and serious symptoms are either already present or not far off. Thus, in a case of aortic disease in which there may be enormous increase in the cubic capacity of the left ventricle, at the bedside he does not think of 'dilatation' as long as the capacious chamber is emptying itself at each systole. This recognition of the difference between what might be called pathological in contradistinction to clinical dilatation assists the student in unravelling the history and symptomatology of hypertrophy with dilatation.

Etiology.—Dilatation of the heart may be *acute* and *sudden* when from any cause the integrity or strength of the muscular

fibres is diminished, as in the various degenerations already described as the fatty, fibroid, &c. It is, however, more likely to follow the form of change known as cloudy swelling or parenchymatous degeneration often associated with the poisoning of the system in severe fevers, as typhus, typhoid, scarlatina, and pneumonia. Anæmic conditions as chlorosis, leukæmia, chronic alcoholism, &c., act in the same manner; but perhaps the most serious cause is the rapid impairment of the cardiac fibre induced by influenza and diphtheria. In acute endocarditis or pericarditis, especially the latter, or when both exist together as in rheumatic fever, acute dilatation may supervene independently of valvular or mechanical causes. In these cases of intrinsic dilatation there may be sudden distension of the cavities, with normal blood-pressure, whilst in the slowly progressive fibroid and fatty degenerations the normal pressure may also bring about the same result gradually. The term *passive* dilatation is sometimes applied to the condition when there has been no antecedent hypertrophy, but it is scarcely applicable to the acute condition.

The most common cause of dilatation is some form of mechanical obstruction slowly produced either in the valves or blood-vessels. This first produces hypertrophy, which brings about compensation, often afterwards followed by dilatation. The mechanism by which this sequence of events occurs will be discussed when dealing with hypertrophy; it is sufficient here to remark that every factor which induces hypertrophy of the heart—viz. valvular, pulmonary, and vascular obstruction, prolonged strain, over-action as in Graves' disease, adherent pericardium, Bright's disease, and fibroid degeneration—is prone afterwards to be followed by dilatation.

Some of these obstructive causes are, however, liable to produce acute dilatation when they operate suddenly, before there is time for enlargement or hypertrophic change in the cardiac muscle. This is more likely to happen when the increased endocardial pressure which the obstruction produces is brought to bear upon muscular fibres already impaired by myocardial degeneration. Muscular exertion, when prolonged and continuous, leads to hypertrophy; but a sudden muscular strain, as in lifting a heavy weight, running up a long flight of stairs, or climbing a steep mountain ascent, often causes acute heart-strain followed by sudden distension of the right side of the heart, and unless the safety-valve action of the tricuspid be sufficient to permit of free regurgitation of the venous blood into the auricle, sudden death may follow. The writer was informed by a physician accompanying a mountaineering expedition which entailed very severe and prolonged exertion in a very high ascent, that obvious signs of great dilatation were observed on examining the hearts of all

the trained guides and of the athletes who accompanied them.

One of the most marked types of cardiac dilatation is seen in the spring months in cyclists who start upon a long ride after comparative inaction during the winter, when the heart-muscle is 'out of training.' In some of these cases under the writer's observation the dilatation has remained for many months, and in a few it has never disappeared, leaving the patient more or less permanently crippled and liable to dyspnœa on slight exertion, constant palpitation, &c., and with an apical systolic murmur.

Of the same nature is the acute dilatation which sometimes supervenes upon acute pleural effusion, œdema of the lung, double pneumonia, and some forms of suffocative bronchitis.

Even in the old-standing cases of great hypertrophy following perfect compensation, the symptoms of dilatation, when they occur, may make their appearance with startling suddenness. The reserve power of the ventricle—that is, the surplus power which the cardiac muscle should possess in order to meet unusual demands over and above what is necessary to carry on the circulation in tranquillity—in these cases has probably been on the wane. Some slight exertion, a fit of severe coughing, or an attack of bronchial catarrh may then be sufficient to disturb the balance; or a smart febrile attack, the supervention of influenza, even with constitutional symptoms so mild as to be overlooked, or some profound mental emotion, may be enough to upset the delicate adjustment. Failure of compensation in these cases may thus be suddenly ushered in with severe symptoms caused by distension of the chambers of the heart without the usual premonitory symptoms. There may be, therefore, in any given case three etiological factors all operating at the same time in producing symptoms of acute dilatation of a heart which is unable to effectually empty its chambers—viz.: (1) Increased blood-pressure within the chamber caused by the original valvular obstruction or incompetence. (2) Degenerative changes in the myocardium leading to impairment of resistance. (3) Extensively increased capacity of the chamber or chambers induced a long time previously by the original mechanical regurgitation or stenosis. In some rare cases, where symptoms of cardiac dilatation have been prominent during life, no mechanical obstruction or degenerative change has been found after death to account for the condition. Such cases are called idiopathic.

Morbid Anatomy.—The appearances necessarily vary with the chamber or chambers affected. When the condition is more or less general, the heart approaches the globular form. The right heart is usually more extensively dilated than the left, for reasons explained in the article on 'Hypertrophy.' In aortic regurgitation all the chambers have their capacities

greatly increased, and in long-standing cases the walls may be found thinned out, though at an earlier stage they had been much thickened. In valvular lesions occurring in subjects addicted to alcoholism, the walls may be very thin owing to absence of any degree of hypertrophy, and in extreme cases the left ventricular wall may be found near its apex to measure little over one-eighth of an inch in thickness. Simple dilatation of the heart, in which its cavities are much distended, probably is never seen in conjunction with a normal thickness of their walls ; some hypertrophy is invariably present when the walls appear normal. Even when the walls are greatly thinned over the distended cavities, hypertrophy is the rule, since the entire organ weighs more than the normal heart.

Where death has occurred in acute cases from strain or other cause there is no hypertrophy, and owing to *post-mortem* contraction the walls may seem of normal thickness. In pure mitral stenosis the left auricle is greatly dilated and may contain more than twice its normal charge of blood, whilst the left ventricle is atrophied.

Dilatation, when extensive, is always associated with marked incompetency of the mitral and tricuspid valves, which may be totally independent of any valvular disease, the auriculo-ventricular rings being usually much widened. Microscopic examination will reveal evidence of degenerative changes when these are the cause of the dilatation.

The changes in other organs may be marked, and venous engorgement of the lungs, liver, spleen, and kidney are usually present.

Symptoms.—In the ordinary *chronic* form of dliatation the symptoms and signs are those of failing compensation, to be described in the article on valvular disease.

In *acute* cases the symptoms and signs are those of rapid failure of the cardiac muscle. The pulse is feeble, small, quick, and irregular ; there is dyspnœa, with coolness of the surface and extremities, and cyanosis. Palpitation and some cardiac pain, with precordial distress and syncopal attacks, are usually present. When the condition follows acute fevers, anæmia, and sudden muscular strain, the sudden over-distension of the cavities may rapidly lead to a fatal syncope before the usual signs of venous engorgement and dropsy have time to show themselves. The phenomenon known as heart-block will be described later on.

The physical signs are characteristic. The apex-beat on inspection is seen to be diffuse ; often it is invisible ; sometimes it is visible, but not palpable ; always it is markedly weak and only to be located with some difficulty. It is displaced outwards and slightly downwards. When the left ventricle is involved a wavy diffuse impulse may be made out in the axillary line, the dulness on percussion corresponding. When

the right side of the heart is distended the impulse and dulness, though also extending beyond the nipple, are markedly increased in the inward direction, so that the flickering wavy pulsation may be observed in the upper part of the epigastrium below and to the right of the xiphoid.

Owing to the feebleness of this epigastric pulsation, it is often quite impossible with the application of the finger-tips, or of the whole hand, to feel it. A much larger portion of the heart is in contact with the chest-wall, but it is right ventricle, the real apex not coming forward; its pulsation is not visible or palpable towards the left, whilst the cardiac dulness always extends considerably beyond the extreme left limit of visible pulsation. An undulatory movement may be observed near to the sternum on the left side in the lower intercostal spaces, and in extreme distension of the right side of the heart this irregular wavy pulsation may be visible as high as the third interspace when there is great distension of the right auricle. The presence of emphysema or obesity will obscure these signs.

The heart-sounds, especially the first, are notably altered even when they are not accompanied by murmurs. The first sound is clear and sharp though weak; it is a short sound resembling the second, and it lacks the booming character heard in simple hypertrophy. Even when there has been no valvular disease, a mitral and a tricuspid murmur may be heard during systole, owing to dilatation of the orifices. That these murmurs are the result of true regurgitation is proven by the facts that the mitral murmur is conducted outwards towards the left and may be heard close to the spine, whilst the tricuspid bruit is accompanied by visible pulsation of the veins in the neck.

In these primary non-valvular cases another sign is usually present, indicative always of grave loss of motor power in the heart. It is usually also present in the valvular cases, and is known as the *bruit de galop*. This triple rhythm sound is difficult to describe, but when once heard is not easily forgotten; it reminds the writer of a cry heard about the promenade of Brighton when repeated a number of times rapidly—'Dyke and back,' the accent being upon the first and last words. Graham Steell is probably correct in regarding it as pathognomonic of failing cardiac muscle, and in his explanation of its production he recognises one sound as being systolic, and the other two diastolic though not caused by reduplication of the second sound. Sometimes the rhythm of the heart-sounds in non-valvular cases resembles that of the fœtal heart or of the ticking of a watch, each sound and the pause between them being of about the same duration.

Where dilatation has supervened upon the hypertrophy of old valvular disease the original murmurs may still be heard, though they are usually weaker; sometimes, however, they

disappear, to return if the heart-muscle improves under rest and treatment. Upon the other hand, the regurgitant murmurs in the non-valvular cases may disappear entirely after the recovery of the cardiac fibre, if this has not been strained beyond its utmost limit.

Diagnosis.—The serious mistake of confounding dilatation with *hypertrophy* is not likely to occur in the presence of the obvious cardiac weakness and embarrassment always present in the first-mentioned condition. The strong and slow heaving impulse lifting an increased but sharply defined area of the thoracic wall outside and considerably below the nipple in hypertrophy, is in striking contrast with the diffuse, feeble, wavy, and irregular impulse of dilatation.

But the problem seldom resolves itself into such simple elements, for, as already stated, the majority of cases met with are those in which both conditions exist together at the same time, and the difficulty in diagnosis is to estimate as far as possible the relative amounts of hypertrophy and dilatation present. The character of pulse and apex-beat, and especially the physical signs of venous or pulmonary engorgement, dropsy, cyanosis, hepatic enlargement, &c., all afford valuable aids.

The increased dulness of *pericardial effusion* is pear-shaped or triangular, and it has often been mistaken for that of dilatation of the right ventricle. Since the fluid in the sac raises the impulse into the fourth or even the third space, the position of the apex-beat should determine the question easily; but too often there is no apex-beat visible or palpable in pericardial effusion, unless the patient be turned upon his face. A careful mapping-out of the limits of dulness, especially in the upward direction on the left side, where it may extend to the second cartilage, and the recognition of the dulness found at the left infrascapular angle, especially if the upper limit of dulness shifts on change of position, are points in favour of effusion which assist in the differential diagnosis.

The dulness of a *pleural effusion* which displaces the apex beyond the left nipple is easily determined by percussing the right side of the thorax; but the presence of an *aneurism* about the root of the aorta, especially if accompanied by some effusion into the pericardium, can only be diagnosed with the greatest difficulty from cardiac dilatation.

Prognosis.—This is always serious, but much will depend upon the cause. In acute cases arising from heart-strain or during fevers, though the prognosis is grave, if the patient rally from the immediate shock of the distension of his heart, he may ultimately recover. In old valvular lesions the prognosis of the supervening dilatation is unfavourable, but by no means hopeless. Under proper rest and treatment many of these patients may have compensation restored, and they may be enabled to live in comparative ease for several years.

In the dilatation occurring in subjects who do heavy manual labour and who take beer or alcohol in considerable amounts, if there be not also marked arterio-sclerosis or degeneration present, the prognosis is fair when the patients' habits can be altered and their lives regulated by proper health laws. The distension of the chambers of the heart so often noticed in soldiers after long marches, and known as the irritable or soldier's heart, generally yields to prolonged rest. In Bright's disease, where the peripheral resistance has caused considerable cardiac hypertrophy, once dilatation supervenes the outlook is most grave.

Treatment.—This, as a rule, is the treatment for failing compensation in old valvular cases—*i.e.* rest, with cardiac tonics. In acute cases *absolute* rest is of greatest importance if life is to be saved ; as there is not time for the action of digitalis or strophanthus, the first measure should be the hypodermic injection of $\frac{1}{10}$ gr. strychnine. Venous engorgement, if great, should be relieved by a free opening in one of the veins of the arm ; if the symptoms be not pressing, a full dose of any saline purgative will act in a similar way by rapidly withdrawing a large amount of fluid from the blood.

Digitalis or strophanthus, combined with strychnine, may be kept up for long periods. The primary cause must be sought out in all instances, and be dealt with on recognised hygienic and therapeutic lines.

HEART, FUNCTIONAL AFFECTIONS OF

As implied in the title, the cardiac disorders included in this group are independent of structural change ; hence they are usually described under the heading of 'Cardiac Neuroses.' Though they are to be regarded as disturbances of function generally occurring in longer or shorter paroxysms, all of them are also commonly met with in association with obvious structural change, as in valvular disease, hypertrophy, dilatation, and degeneration, but especially during the stage of failing compensation. The functional disorders of the heart may be classed as sensory and motor, but both varieties of affection may exist together. Angina pectoris, especially of the pseudo or spurious type, may be taken as illustrating the sensory variety, and tachycardia may be regarded as a purely motor disorder of which the patient may be entirely unconscious.

PALPITATION OF THE HEART

This is a form of functional disorder in which the sensory and motor disturbances are blended, as the essential feature in it is a consciousness of the increased force of the cardiac action

amounting to discomfort, to distress, or to severe pain ; often the abnormal over-action of the heart is associated with irregularity, but not necessarily so. As this is the most frequent form of functional disturbance, it may be briefly described first.

Etiology.—The condition is much more common in women, in whom it is often observed at the menopause and at the menstrual periods. Being a pure neurosis, as might be expected it is an almost constant element in the clinical picture of hysteria. The immediate cause of the attack is often some mental excitement, shock, or emotion or violent exercise, acting directly upon the nervous mechanism of the heart. Dyspepsia acts reflexly, and is a very common cause, especially when associated with the production of much flatus in the stomach or intestines. Toxic substances, as tobacco, tea, coffee, the poison of gout, and alcohol, are powerful etiological factors, as are also all agents which tend to produce nervous exhaustion, as sexual excesses, anæmia, irregular living, or dissipation.

Palpitation has been unfortunately raised to the dignity of a 'disease' under the title of the 'irritable heart of soldiers,' where, however, it is but a symptom of mechanical strain which has produced a degree of more or less permanent dilatation. The *irritable heart of young persons*, the direct result of irregularities in diet and in exercises, both physical and mental, is a purely functional disturbance, graphically and forcibly described by Professor Allbutt in his brilliant article in the 'System of Medicine,' vol. v.

That attacks of palpitation may be produced by agents which act upon the vaso-dilator mechanism is highly probable, since the influence of amyl and other nitrites shows itself in tumultuous and often distressing cardiac action, which appears sometimes to be closely allied to the functional disturbance seen at the climacteric period.

It is mere speculation to discuss the method by which the increased cardiac action is produced—viz. whether it be vagus paralysis or stimulation of the accelerators, &c.

Symptoms.—The attack may last but a number of seconds, the patient being startled by a few violent thuds of the heart against the chest-wall, which are obviously caused by some transient mental excitement. More frequently, however, the attacks last for several minutes, and sometimes for hours, coming on in paroxysms, during which there are all degrees of cardiac discomfort, from an unpleasant sensation of fluttering to one of more or less anxiety associated with real pain and violent throbbing or over-action of the heart, with visibly increased pulsation of the large vessels. The pulse may quicken to 150 or even to 200, and there may be cerebral symptoms, as vertigo, tinnitus, and throbbing in the ears,

probably the result of dilatation of the arteries ; combined with these are mental phenomena caused by anxiety and dread of the effects of the attack. There may be flushing of the face or pallor, according to the increase or diminution of the blood-pressure caused by the tightness or slackness of the vascular walls. The attack may pass off as suddenly as it came, or it may gradually subside, leaving behind it a feeling of exhaustion or nervous debility. Sometimes the paroxysm is cut short by the patient moving about actively, and usually in neurasthenic subjects there is an abundant discharge of pale urine immediately after its cessation.

In dyspeptic patients immediate or partial relief is experienced on the eructation of large quantities of gas from the stomach. It will be noticed that all the above symptoms are commonly met with in organic heart-lesions ; but these, along with angina pectoris, are excluded in the consideration of the present article, which only deals with palpitation as a functional affection.

On examination, the heart-beat is visible in its normal position, but to the eye and touch the ventricular contractions are obviously greatly exaggerated. There is marked pulsation in the large vessels, and occasionally the entire precordial region heaves with the increased cardiac movements. The sounds are abnormally clear and ringing, the second one being much accentuated and even metallic in character at the base. Murmurs are absent, unless the patient be markedly anæmic, and the area of cardiac dulness is usually normal. The pulse, beyond the increase in its rate, may give no indication of the increased force expended by the ventricles.

There is occasionally to be met with a curious sensory disorder of the heart, which, for want of a better name, is called *false palpitation*, in which there are no physical signs, the symptoms being entirely subjective. The patient complains of severe and painful palpitation, though the heart is apparently acting quite normally. This condition is almost always met with in subjects of the hysterical or neurasthenic type.

Diagnosis.—This resolves itself into the exclusion of organic disease, which, as a rule, is not difficult when an opportunity is obtained for examining the heart in an interval of quiet. In the attack, when the patient has not been seen previously, the presence of a functional systolic apical murmur is liable to mislead. The character of this bruit is soft, its duration short, and the first sound can usually be distinguished clearly, notwithstanding the presence of the murmur, and there are no physical signs indicative of hypertrophy or dilatation.

A point of considerable diagnostic value may usually be gleaned from the history of the case : often the attacks have

occurred during the night, arousing the patient from sleep with all the distressing sensations of nightmare.

Prognosis.—This is always good when the diagnosis of absence of organic disease has been settled, and when the patient can be positively assured of the functional nature of the ailment.

Treatment.—During the attack, rest in the recumbent position with the head slightly elevated, the administration of a carminative or a diffusible stimulant, and a calm assurance of the absence of danger, are all that is required. When the paroxysm is evidently the result of dyspepsia, especially when organic acidity is a prominent feature, a large dose of sodium bicarbonate with oil of peppermint will afford relief; vomiting sometimes will cut short the attack in these cases.

In the interval between the attacks, careful attention must be paid to hygienic details, as diet, exercise, sleep, rest, &c.

As regards drugs, much harm is often done by the haphazard prescribing of powerful agents like aconite, amyl, digitalis, belladonna, and strychnine, which are liable to disturb seriously the delicately adjusted mechanism of the heart. Perhaps the best routine treatment is a nerve-tonic like Easton's syrup, in small doses, combined with a few minims of tincture of digitalis or of strophanthus. Strychnine in full doses always increases the palpitation of hysterical subjects. Night attacks are usually warded off by a moderate dose of bromide. A month's course of iodides is often of great value.

The motor disturbances of the heart of functional origin are increased rapidity or tachycardia, diminished frequency or bradycardia. and alterations in rhythm or arrhythmia.

TACHYCARDIA

This term is not synonymous with rapid action of the heart such as is found in all inflammatory and feverish conditions and in many other diseased states. It is a paroxysmal disorder, the pathology of which is probably that of a cardiac neurosis allied to the condition which is observed in exophthalmic goître. The name 'tachycardia' is, however, often used to include two distinct states which cannot be considered as of the nature of functional cardiac affections. These may be mentioned with the intention of excluding them from the typical paroxysmal neurosis to be presently described; they are: (1) Those rare cases where the normal pulse of the patient has been *always* rapid; some examples of this condition have been recorded where the pulse was persistently over 120 throughout life; (2) cases where tumours have caused pressure upon the pneumogastrics or where the nuclei of these nerves have been involved in bulbar paralysis. When these cases, together with those showing rapidity of pulse in organic

heart-affections, in ordinary palpitation, and in Graves' disease are excluded, there remains the symptom-complex to which the term 'quick heart' or 'tachycardia' can be correctly applied.

Etiology.—The disease is found in both males and females in about equal frequency; it has been noticed in childhood, but usually it first appears about middle-life. Neurasthenia, heart-strain, toxic causes, as those arising from intestinal putrefaction, mental shock, reflex causes as ovarian disease, floating kidney, &c., have all been supposed to be etiological factors.

About the pathogeny of this diseased condition nothing whatever is known; its resemblance to the physiological effects of atropine-poisoning on the heart suggests that it may possibly be the result of some ptomaine, but whether the result of stimulation of the accelerators or paralysis of the vagi, either centric or peripheral, or of affection of the cardiac ganglia or muscle or cerebral cortex, is a subject only of speculation. The condition has no morbid anatomy, though, when of long continuance, slight dilatation has been observed.

Symptoms.—The attacks, as already stated, come on paroxysmally; hence the disease has been called by Williams paroxysmal tachycardia, a nomenclature rejected by Allbutt, because true tachycardia always exhibits itself in this way: With little warning, and often with no consciousness of precordial discomfort, or even of palpitation, the pulse-rate suddenly rises to 200 or more per minute, as counted by the stethoscope. The paroxysm may last for a few hours or for several days, in which case symptoms of dilatation may be observed, or of venous engorgement, dropsy, and albuminuria. Usually, however, the attack ends as suddenly and abruptly as it commenced.

In some cases painful sensations, as a sense of tightness or constriction about the heart, may be experienced, but often, unless in protracted cases, the patient may be able to move about without inconvenience or breathlessness, and sometimes the rapidity of the ventricular contractions has only been discovered accidentally. Sleeplessness is usually complained of, but when sleep does supervene the pulse continues to run riot as before.

Diagnosis.—The paroxysmal or intermittent nature of the diseased condition excludes those cases of quick pulse which are caused by pressure on the trunks or disease of the nuclei of the vagi, and those instances of naturally quick pulse. *Organic heart-lesions*, as mitral obstruction and dilatation, when they produce great rapidity of the cardiac contractions, are always associated with marked irregularity, or arrhythmia; whilst in tachycardia the rhythm is always regular, though the pulse-rate may reach 250.

Tobacco-heart, when very frequent, is always irregular, and the same is true of the pulse in the third and fourth stages of digitalis-poisoning, and of the pulse of the *alcoholic heart.*

Exophthalmic goître, before the advent of the prominence of the eyeballs and muscular tremor, simulates tachycardia, but the quickness of pulse is continuous and not intermittent, and there is always marked throbbing of the vessels.

In tachycardia, though the rapidity of the contractions is so enormously increased, the output is not really augmented ; hence, even in prolonged or frequently recurring attacks, hypertrophy does not take place.

Prognosis.—Death from syncope is liable to occur in elderly patients when the attacks are prolonged or of very frequent occurrence. In young subjects the outlook is good, though a complete cessation of attacks is not often noticed ; the condition may last till long past middle-life, after which the attacks may be separated by very lengthened intervals. According to Herringham, if attacks of tachycardia continue to occur after the age of thirty, the patient is not likely to survive the age of fifty.

Treatment.—Beyond rest, little can be done to relieve the paroxysm, though some patients resort to various manœuvres which tend to slacken the speed of the cardiac revolutions. Thus compression of the thorax and abdomen, with powerful flexion of the thighs upon the abdominal walls, is sometimes successful. Cardiac tonics are generally useless ; but in the intervals between the attacks, in small doses, combined with arsenic and iron, they are of undoubted value. Sansom advocates a weak continuous current applied over the vagi, and in mild cases this is beneficial. Pressure over both vagi may be tried, and ice has been applied with some benefit over the heart.

BRADYCARDIA

The term indicates slowness of pulse, and, according to Allbutt, nothing more, as it does not designate a true symptom-group, though Morison regards it as a '*disease*' of middle and later life. The paroxysmal type of bradycardia will be described separately.

Etiology.—Whether regarded as a symptom or a disease, it is met with much more frequently in males. Amongst the causes of the condition (which should not be made to include those rare cases where the pulse is naturally persistently slow) the following are believed to be operable : cardiac overstrain ; syphilis ; degeneration of the cardiac fibre, either fatty or fibroid ; aortic-valve disease ; toxic agents, as lead, alcohol, muscarine, tobacco, digitalis, opium, and the poisons generated in uræmia, jaundice, and diabetes ;

the exhaustion produced by the various fevers, especially influenza, diphtheria, typhus, &c. In these latter cases, bradycardia appears during convalescence. A very important group of causes is that where there is disease of the stomach, as dilatation, ulcer, cancer, &c. Pain arising from affections of a large nerve like the sciatic causes marked slowing of the pulse.

Bradycardia is a common phenomenon in cerebral apoplexy and tumour, and various mental states as melancholia, hysteria, epilepsy, general paralysis, &c.; it occurs also in affections of the upper portion of the spinal cord. Most conditions associated with a marked rise of blood-pressure will slow the pulse, as is often seen in capillary contraction. After parturition the pulse sometimes slows very considerably.

The explanation of simple bradycardia is difficult; it differs very materially from the opposite condition of tachycardia in being so frequently associated with cardiac and vascular degeneration, and hence it is very doubtful if it should be included under functional heart-affections. Some cases, especially the toxic group, are probably the result of stimulation or irritation of the vagi, peripheries, or nuclei; others are supposed to be due to paralysis of the accelerators or of the intracardiac ganglia. When the slowing of pulse is caused by organic heart-lesions, arrhythmia is usually associated with it. The pathogeny will be further discussed in the description of the paroxysmal form of bradycardia.

Symptoms.—Little need be said under this heading, save that in acute transient cases the pulse may suddenly fall from the normal to forty, thirty, or even twenty beats, when syncope may supervene. Such attacks must be regarded as evidence of cardiac failure, and if they return or remain for any length of time death from syncope is almost certain to wind up the case. Morison regards the fatal form of such attacks as of the nature of a recurrent syncope.

Diagnosis.—The condition is characterised by the one symptom of slowness of the pulse, but one very important consideration must not be overlooked. The cardiac revolutions should always be counted at the heart by the aid of the stethoscope, as a feeble ventricular systole may not be perceptible at the wrist Dehio suggests that by the administration of atropine a diagnosis can be made between bradycardia the result of intrinsic cardiac causes and that which is the result of causes outside the heart, since in these latter the heart can be accelerated by the drug. J. Mackenzie has recently drawn attention to the importance of studying the action of digitalis from a similar point of view, and he has demonstrated the production of a minor degree of heart-block by administering the drug.

Prognosis.—In the presence of serious organic disease the forecast is grave, while in acute toxic cases it is hopeful; in

other words, the prognosis of simple bradycardia is that of the diseased condition with which it is associated.

Treatment.—The aim should be at the removal of the cause when this is possible. Cases depending upon the exhaustion following fevers and toxic agents yield to strychnine hypodermically, followed up by small doses of cardiac tonics of the strophanthus type.

BRADYCARDIA, PAROXYSMAL

This condition is also known as the Stokes-Adams Syndrome; and since heart-block is a common event during the progress of the disease, by some authorities the title of 'Stokes-Adams disease' is regarded as practically synonymous with 'heart-block.'

The form of bradycardia under consideration is characterised clinically by slowness of the pulse, which, though usually permanently infrequent, becomes still slower during the attacks; by cerebral disturbances of a vertiginous, epileptiform, or apoplectiform character; by syncopal attacks and by systolic pulsation of the cervical veins synchronous with the auricular contractions.

Etiology.—The majority of the causes already mentioned under the etiology of simple bradycardia may be factors in the causation of the paroxysmal variety. The disease seldom appears till after the age of fifty. Arterio-sclerosis, fibroid and fatty changes in the cardiac muscle, syphilis and old valvular lesions, are recognised factors. A variable proportion of the cases originates in the toxæmia following influenza, typhoid fever, diphtheria, and other infective processes. Some examples of the disease undoubtedly are of the nature of a true neurosis independent of structural change in the heart or elsewhere; whilst in a small number the affection appears to be the result of organic disease within the cranium, involving the vessels supplying the pneumogastric centres.

Pathogeny.—The subject of *heart-block*, which is intimately associated with the pathology of bradycardia, has attracted considerable attention during the past few years, mainly through the influence of the researches of Gaskell, Englemann, Hering, Aschoff, Mackenzie of Burnley, G. A. Gibson, and others. Tawara, by the most painstaking examination of 112 hearts removed from patients who had exhibited marked cardiac feebleness during life, was able to demonstrate the absence of inflammatory myocardial mischief in the great majority of them, even when old valvular lesions were present. The view is now freely accepted that heart-block is the outcome of a defect in the auriculo-ventricular bundle of muscular fibres associated with the name of His. The function of this muscular bundle is obviously to convey the stimulus for contraction from the auricles to the ventricles. The bundle

of fibres, according to Tawara, begins in the neighbourhood of the anterior edge of the coronary vein, and then passes forwards on the right side of the auricular septum below the foramen ovale, lying close upon the auriculo-ventricular septum. It forms a special knot-like thickening, consisting of an intricate mass of muscular fibres immediately above the point of insertion of the median flap of the tricuspid valve. From this nodule muscular processes are given off which are eventually distributed under the endocardium and form a lining for the entire inner surface of the ventricular cavities, fusing everywhere with the ordinary fibres of the cardiac muscle and entering into the anterior and posterior papillary muscles after traversing the trabeculæ. Tawara has been able to trace these conducting fibres from their origin at the mouth of the coronary vein to their ultimate termination in the ventricular walls by observing the small amount of sarcoplasm which they contain, and by noting their tendency towards reticular formation, interlacement, and fusion, which contrasts markedly with the behaviour of the ordinary cardiac fibres. This observer has also established the fact that the mysterious fibres discovered and described half a century ago by Purkinjie are identical with the main branches of the ventricular part of the auriculo-ventricular bundle.

Hering and Erlanger have demonstrated the important fact that, by causing compression of the fibres of this connecting auriculo-ventricular bundle of His, the condition of heart-block can be produced at will. When the pressure is so slight as not to stop completely the transmission of the stimulus from auricle to ventricle, an occasional arrest of the ventricular systole occurs, or the ventricle responds to only every second, third, or fourth auricular contraction. When the block in the transmission of the stimulus is complete, the auricles continue to beat at their normal rate, whilst the ventricles exhibit an independent speed of their own, which is estimated to be not more than about thirty beats per minute.

In several instances the fibres of the bundle of His have been found altered by diseased processes—rheumatic, gummatous, tubercular, fatty, sclerotic, &c.—in hearts which, during life, had exhibited the phenomena recognised as the Stokes-Adams syndrome. It is probable, as suggested by Aschoff, that the block is complete when the knot-like nodosity or the main branches are diseased, and that changes in the smaller processes only cause a minor degree of blocking of the conduction of the stimulus from the auricle to the ventricle.

The bundle of His does not undergo enlargement in hypertrophy of the heart the seat of valvular disease, and the last-mentioned authority believes that this fact may explain the gradual failure of the ventricle, owing to the bundle remaining too small in proportion to the greatly increased

bulk and number of the ordinary cardiac fibres. The subject of heart-block, and the methods of recognising affections of the auriculo-ventricular conducting fibres, are ably set forth by J. Mackenzie in the 'British Medical Journal' of October 27, 1906.

Symptoms.—As already stated, these consist of slowing of the pulse, which may be constantly present, but which is generally exaggerated during a paroxysm. In one case the writer witnessed a drop to eighteen beats in the minute; but less than half this number of beats has been recorded. Arrhythmia of various types may be present when the block is only partial. Breathlessness is often marked, and sometimes cyanosis. Syncopal attacks are common, but total stoppage of the ventricular contractions only occurs when the block is complete.

The total arrest of the ventricular action accounts for the cerebral symptoms, since it has been demonstrated that the pseudo-apoplectic and epileptiform seizures are preceded by the arrest of the ventricular contractions. Cheyne-Stokes and other disturbances of respiration may be occasionally observed.

Many observers, including Gibson and Mackenzie, have demonstrated by graphic methods that the pulsations in the veins may be six or seven times more frequent than the arterial beats. Upon careful auscultation, feeble sounds can be heard in the intervals between the ventricular contractions, and in the hands of Magee Finny, and Ritchie the fluorescent screen has demonstrated the contractions of the auricles during these intervals, in which the ventricles are in complete repose.

Diagnosis.—The different points have been already emphasised, and do not require repetition. The main problem is the recognition or detection of the nature of the primary cause of the blocking of the impulse or stimulus between the auricles and ventricles. Osler remarks that in young adults and middle-aged men syphilitic myocardial changes are probably the cause of the condition.

The prognosis and treatment are those of the primary state; and since this is so frequently an advanced senile change little can be expected from drugs. Rest, strychnine, and iodides, in the present state of our knowledge, afford the best hope of a prolongation of life. The danger of a complete arrest of the heart's action setting in suddenly must never be lost sight of.

ARRHYTHMIA

As the term implies, this functional affection of the heart includes all those cases where the cardiac rhythm is disturbed; it excludes those conditions where the speed of the

heart is simply increased or diminished. *Irregularity* as to speed and force is an essential feature in the condition under consideration, but it includes also *intermittence.* Though this latter is a deviation from the normal rhythm, it may nevertheless present a rhythm of its own which may be marked by striking regularity.

Etiology. — The condition has been found at an early age, but most frequently it appears after middle-life, and it is more common in males. Tobacco answers for a considerable number of the cases met with in ordinary practice ; tea, coffee, gout, mental shock, influenza, sexual excesses, the unskilful administration of digitalis, aconite, and other drugs are not uncommon causes.

Reflex arrhythmia is observed in unhealthy conditions of the stomach, and in elderly subjects it is often a symptom of dyspepsia, especially when much flatus is present.

In all the above conditions the irregularity or intermittence may be caused by some interference with the action of the vagus or other nervous mechanism of the heart, without any structural cardiac change being present. It must, however, be always remembered that, like the other neuroses of the heart—palpitation, bradycardia, and tachycardia—the condition may be intimately associated with valvular disease, especially with mitral affections, with degenerative changes in the myocardium, and with dilatation, however caused.

It is often observed in cerebral hæmorrhage and in brain-diseases.

The recent researches in the phenomena of heart-block, described upon page 568, afford strong evidence that many varieties of arrhythmia are due to involvement of the auriculo-ventricular bundle of His.

The entire subject of arrhythmia has been illuminated by the remarkable investigations of Mackenzie of Burnley. He has shown that the continuous irregularity of the heart found so frequently in mitral disease, especially in mitral stenosis, and also in the senile state associated with arterial sclerosis, is due to *abnormal inception of the cardiac rhythm.* Half of the cases of serious cardiac failure illustrate this interesting phenomenon.

As the result of these recent researches, Mackenzie maintains that the normal sequence of the heart's contraction no longer prevails in such cases, the ventricles alone contracting, or the auricles and the ventricles contracting simultaneously. In the normal condition, the stimulus for contraction arises at the mouth of the great veins in the sinus-node at the root of the superior cava, and passes downwards from the auricles to the ventricles. In the abnormal condition under consideration, the starting-point of the heart's contraction is located in the nodal swelling on the auriculo-

ventricular muscular bundle of His, which is situated at the line of junction of the auricle with the ventricle. The result of this abnormal inception is irregularity ; the auricular systole, which previously preceded the ventricular contraction, disappears, and the auricles and ventricles are then found to contract at the same time.

The irregularity so produced is characterised by the absence of the orderliness observed in other forms of arrhythmia. According to the observer just quoted, long and short cardiac cycles appear in the most disorderly fashion, so that he applies to this variety of irregularity of the heart's action the term *disorderly rhythm*. The above conclusions regarding the existence of auricular systole are drawn from a long series of observations recorded by various graphic and clinical methods which demonstrate—(1) the presence of a wave in the jugular pulse-tracing and in the liver-pulse, and a presystolic tricuspid murmur due to a contraction of the right auricle ; (2) a wave in the apex-tracing and a presystolic murmur due to contraction of the left auricle.

The result of the abnormal rhythm at once seriously embarrasses the heart in carrying on its work. If the muscular substance be in good condition, the disturbance may pass off under digitalis and rest-treatment ; but if advanced degenerative changes exist, a fatal issue may speedily follow in spite of all treatment. Mackenzie is led to regard the function of the auriculo-ventricular bundle of His as not merely one of conduction, but he believes that it possesses also the power of creating a stimulus.

Symptoms.—The intermittent form of pulse is characterised by an irregularity which shows itself in a dropped beat, occurring after a number of normal and regular pulsations. This intermittence may follow every third or fourth beat, or it may only occur once or twice in a minute. The stethoscope sometimes reveals a feeble ventricular systole which is not represented at the wrist. All sorts of irregularity may be noticed which are caused by variations in the intervals between successive beats, though each beat may in every respect resemble its fellow. These *irregularities of time* rarely exist without some *irregularities in the force and character* of the ventricular contractions or pulse-waves, and the latter condition is of more serious omen than mere intermittence.

The *pulsus bigeminus* and the *pulsus trigeminus* are caused by two or three pulsations rapidly following each other, and separated from the next two or three beats by a comparatively long interval. The phenomenon is not, however, a mere time-irregularity caused by variations in the length of the interval between the beats, since the first beat is usually stronger, being caused by a hypersystole, and the second or third by a hyposystole ; and the long pause may denote a

condition of asystole of the heart. These conditions are usually associated with mechanical obstructions, as mitral disease, but they may be produced by digitalis.

The *pulsus alternans* is one in which a strong and a weak systole alternate with each other, producing a full radial beat, followed by a feeble one which may be perceptible with difficulty.

The *tic-tac* rhythm characteristic of the fœtal heart is rarely found in purely functional heart-affections, whilst common in the advanced dilatation of failing compensation.

The serious modification of rhythm mentioned on page 559, and known as *galloping* rhythm, is always an indication of failing muscular power, as is also the condition known as delirium cordis, in which irregularities in time and force reach their maximum, and the condition known as heart-block (page 568) when partial may be associated with arrhythmia.

Often the arrhythmia is entirely an objective sign, the patient being unconscious of any sensation of irregularity; but at times the dropped beat causes a distressing feeling as if the heart was about to stop finally, and it is worth remembering that as a rule the irregularities which attract the attention of the patient are of less gravity than those of which he is entirely ignorant, and they are usually associated with some palpitation.

Intermittence may last for many years, but even when chronic it is liable to entirely disappear for short periods.

Diagnosis.—The condition itself is so obvious, that diagnosis presents no difficulties, the main problem being the exclusion of organic heart-affections. This is occasionally impossible without watching the case for some time, as, according to Huchard, a soft mitral systolic murmur may ultimately develop in the purely functional cases which, without a history of the illness, might be mistaken for old mitral disease.

Prognosis.—When the cause is a removable one the prognosis is good, as in tobacco-heart and in dyspepsia; but when the condition first appears after middle-life the presence of organic disease, or its supervention at a later period, renders the prognosis serious. Examples of intermission lasting a long lifetime are numerous.

Treatment.—When a careful search has revealed the probable cause, the removal of this is obviously imperative; excessive tea- or coffee-drinking and tobacco-smoking, and every cause which induces dyspepsia, must be avoided. The cases caused by mental shock rapidly improve under rest, and those following muscular strain are probably due to some degree of acute dilatation, and they recover under the same treatment. The treatment applicable to most cases of palpitation is effectual—viz. small doses of strophanthus or

digitalis, with iron, quinine, and strychnine. In the presence of organic disease the remedies suitable for the valvular or degenerative change are indicated.

HEART, HYPERTROPHY OF

This is the condition in which there is enlargement of the organ caused by increased growth of the muscular fibres in the walls of one or more of its cavities. The enlargements of the heart associated with fibroid and fatty degenerations are not included under the term 'hypertrophy' except so far as these changes have led to hypertrophy of the remaining fibres of the muscular tissue. The condition is usually found in association with more or less dilatation, so that in most text-books on medicine the subject is usually treated under the heading 'Hypertrophy with Dilatation'; and since this state is generally secondary to valvular disease, it will be best studied when describing affections of the different cardiac valves. As, however, there are also conditions in which hypertrophy of the heart is sometimes found without any structural alteration in the valves, it is considered advisable briefly to discuss cardiac hypertrophy under a separate heading.

By the term *simple* hypertrophy of the heart is meant the condition in which the muscular walls are increased in thickness, the cavities remaining practically normal in their capacities. *Eccentric* hypertrophy is the condition where with the thickened walls the chambers are more capacious; it is a synonym for hypertrophy with dilatation. *Concentric* hypertrophy is the term employed to define the condition in which, with the increase in thickness of the walls, the cavities are diminished in size. This latter state probably does not exist save as a *post-mortem* change corresponding to *rigor mortis*.

Morbid Anatomy.—The size and weight of the heart may be greatly increased; in rare instances enormous enlargement may be met with, but these very large hearts always belong to the type of hypertrophy with dilatation. Taking the average weight of the male heart as 9 oz., or 255 grms., and its bulk as about that of the clenched fist of the individual to whom it belongs, it is not unusual to find the weight of the enlarged organ to amount to 16 oz., or 453 grms., with a corresponding increase in bulk. A weight of almost three and a-half times this—*i.e.* 53 oz.—is mentioned by Osler as having been reported by B. Robinson.

The shape of the heart will vary with the chambers involved; as a rule it tends towards the globular form. Owing to the widening out or blunting of the apex, which consists mainly of the hypertrophied right ventricle, the so-called 'quadrate' form may be produced. In aortic regurgitation the hyper-

trophy reaches its maximum in the *cor bovinum*, or ox-heart. When the left ventricle alone is enlarged, the heart may retain its conical form. On section the ventricular walls may measure thrice their normal thickness. The hypertrophy is less marked in the more fibrous walls of the auricles. The muscular fibre is firm and offers considerable resistance to the knife and fingers. The papillary muscles, columnæ carneæ, and the musculi pectinati participate in the hypertrophy. The increased bulk of the heart is found on microscopic examination to be owing to two causes—there is an increase in the actual size of the muscular cells, and their number is also increased. It is a very noteworthy fact that the auriculo-ventricular bundle of fibres of His, which will be found described under 'Heart-block' (page 568), does not share in the hypertrophy.

Etiology.—The causes are either *intrinsic—i.e.* those acting inside the heart itself—or *extrinsic*. Of the former the most common is valvular disease; this will produce hypertrophy of the left ventricle when the aortic valves are either stenosed or incompetent, or when the mitral permits of regurgitation. The dilated and hypertrophied condition of the left ventricle causes an increased pressure in the left auricle, with raised blood-pressure in the lungs, to overcome which the right ventricular walls must put forth more force, so that they also enlarge. In this way ultimately both sides of the heart become increased in capacity and in weight.

In *pure* mitral stenosis, however, the increased intra-auricular pressure first leads to dilatation and hypertrophy of the left auricle, then to raised tension in the pulmonary circulation, to overcome which the *right* ventricle must enormously increase to carry on the circulation. The heart, which may reach twice its normal weight, is made up chiefly of right ventricle, whilst the left may be normal or even atrophied. Since, however, the stenosis of the mitral valve usually permits of some degree of regurgitation at the same time, the left ventricle is also generally found to be more or less dilated and hypertrophied.

Valvular lesions on the right side of the heart cause hypertrophy in the same manner as on the left; they are, however, rare as primary affections, being usually secondary to left-heart lesions through relative insufficiency.

Another intrinsic cause leading to cardiac hypertrophy is fibroid degeneration. As already stated, this condition is not to be confounded with true hypertrophy, but the mechanical impediment which it causes to the circulation may ultimately produce hypertrophy of the remaining intact muscular fibres. This may occur even in fatty degeneration or infiltration, but only to an extent which is of no clinical importance.

Adherent pericardium, by embarrassing the heart, may also be regarded as another intrinsic etiological factor in cardiac hypertrophy.

The over-action of the heart in exophthalmic goître which leads to hypertrophy with dilatation, though usually tabulated as an example of intrinsic causation, probably belongs to the next category.

Extrinsic causes which induce cardiac hypertrophy sometimes afford the most perfect examples of the pure affection. Thus, general arterio-sclerosis and chronic Bright's disease, by diminishing the calibre of the capillaries and small arteries, raise the peripheral resistance and thus enormously increase the load in front of the left ventricle. In accordance with the universal law that within certain limits increased muscular work must be followed by increased muscular growth and development, the cardiac muscle responds, and hypertrophy of the left ventricle sets in as a conservative or compensatory process. The heart in arterio-sclerosis may be found to weigh up to 30 oz., all its valves being intact and its chambers little, if at all, dilated. The right ventricle participates to a much less extent than the left, but it is always somewhat hypertrophied. In the same way all toxic agents, as alcohol, lead, uric acid, excessive tea-drinking and tobacco-smoking, and everything which raises the blood-pressure, will tend to induce cardiac hypertrophy by increasing peripheral resistance.

Of still more importance is the increased resistance caused by constant and prolonged muscular exertion in those who follow laborious occupations ; the increased size of the heart of athletes is an illustration of this form of general hypertrophy. The large heart of pregnancy belongs to the same group.

To the extrinsic causes must also be added all those conditions of the lung, as emphysema, cirrhosis, &c., which present obstacles to the pulmonary circulation and lead to hypertrophy of the right ventricle. It is a noteworthy fact, and contrary to what might be expected, that cardiac hypertrophy does not supervene upon aortic or other large aneurisms unless the aortic valves become implicated ; but hypertrophy occurs in cases of congenital narrowing of the aorta.

Symptoms. — Even in advanced cases, where the hypertrophy is secondary to a valve-obstruction, the conservative process has kept pace with the demands made upon the heart so steadily and successfully that the condition of perfect compensation has become firmly established without the patient having become aware of the valve-impediment or of the great increase in the bulk or force of the heart by which nature has adjusted the balance.

Hence the condition is usually entirely latent and devoid of symptoms till compensation begins to fail. The patient may first become aware of the increased power of the enlarged

organ under some emotional disturbance when the apex strikes the chest-wall ; there may be pain and some vague cardiac uneasiness and quickness of breathing, with a sensation of fulness in the head and throbbing, and flashes of light before the eyes and ringing in the ears. These symptoms pass off with the excitement which produced them. They may be called into prominence not only by mental but by muscular excitement, and by any dyspeptic condition which causes accumulation of flatus and elevation of the diaphragm ; excessive tobacco-smoking may induce them.

The above transient disturbance may be taken as the forerunner of signs of failing compensation ; it is probably a warning that the reserve force of the heart is not quite able to meet the demands made upon it under mental or muscular excitement. Gradually the patient begins to notice that he is continually conscious of the ventricular contractions unless he is in a state of complete repose, and with this consciousness is usually associated some degree of pain or uneasiness ; in other words, one of the first symptoms of cardiac failure has developed—*palpitation*. If the patient be seen at this stage, the hypertrophied organ may be found heaving with great force in the precordial region, and there may be slight irregularity in its action.

Much importance is attached to the recognition and interpretation of the condition of affairs at this stage. The signs and symptoms must not be taken as evidence that the hypertrophy has progressed to an injurious or dangerous extent. The fact is that the abnormal degree of cardiac pulsation is not the result of a superfluous or unnecessary over-action of a greatly increased organ, but it should be recognised as an evidence of *weakness*, the hypertrophied heart being unable to perform effectually the increased labour demanded from it.

Cough, occasionally followed by slight hæmoptysis ; *quickness of breathing* on exertion, passing at a later stage into *dyspnœa* ; *œdema* of the legs, and the other signs of dilatation and venous engorgement supervene if the balance be not again restored by rest and judicious treatment.

As already stated, the hypertrophy may involve the entire organ or only one or two of its chambers. The signs indicative of each condition may be briefly considered.

Hypertrophy of the left ventricle.—The symptoms of the *causes* which produce this condition will be present, and need not be here discussed. As already stated, these are : (1) General arterial sclerosis, Bright's disease, and conditions which induce increased blood-pressure ; (2) aortic obstruction ; (3) aortic regurgitation ; (4) mitral regurgitation ; (5) fibroid degeneration ; (6) adherent pericardium ; (7) Graves' disease ; (8) congenital narrowing of, or the pressure of tumours upon, the aortic trunk ; (9) prolonged muscular exertion, as in occupations involving severe labour.

Physical Signs.—Before dilatation and failure of compensation has occurred bulging may be obvious on inspection where the disease has commenced in early life, the intercostal spaces from the third to the seventh being widened and prominent on the left side of the sternum. The apex-beat is visible over a wider area than normal ; but it is not diffuse as in dilatation. The apex strikes the chest-wall with considerable force in the sixth or seventh space and outside the nipple-line ; thus it is, in comparison with the normal beat, displaced downwards and outwards.

On palpation the increased force is felt to be of a slow heaving character ; it may be detected extending often to the eighth space, and as far outwards as the anterior line of the axilla. Emphysema or other condition may obscure the extent and sharpness of the impact. When dilatation supervenes, the apex-beat will be felt diffused and weakened, the slow deliberate heave being replaced by a rapid and abrupt movement, sometimes of a wavy or oscillating character.

Percussion will reveal a marked increase in the area of cardiac dulness, both superficial and deep, especially towards the left side ; but the dulness is increased in all directions, though not equally. It may be found commencing at the lower part of the left second intercostal space, and extending downwards and outwards to the eighth space in the anterior axillary line at the apex-beat ; whilst to the right it may extend for an inch or more beyond the right margin of the sternum. Thus in great hypertrophy of the left ventricle with dilatation there may be, in the horizontal direction, unbroken dulness extending on the right from two inches beyond mid-sternum to the line of the anterior fold of the armpit, or even to the mid-axillary line on the left, and dulness may be even detected behind near to the spine. Auscultation reveals a long, booming, distant, or muffled first sound with marked predominance of the muscular over the purely valvular element of the sound ; the long pause is somewhat shortened, and the short pause lengthened. The aortic second sound is clear and accentuated. When dilatation is marked along with the hypertrophy, the first sound is very distinct and sharp, and the second sound louder.

These qualities of the sounds are absent, or made out with difficulty in the presence of murmurs caused by the original valvular mischief, and they are often obscured in the pure hypertrophy of renal disease by reduplication. Occasionally the first sound heard immediately inside the apex-beat may exhibit a distinct metallic timbre in this latter condition.

The pulse in pure hypertrophy is full, regular, and strong, with increased tension ; under the finger-tips its duration may be made out to be distinctly prolonged, unless the heart is acting rapidly. When a marked degree of dilatation has supervened, the pulse increases in frequency, the tension falls,

and irregularity or intermittence is observed. The peculiar characters of the pulse in aortic regurgitation will be referred to in the description of valvular diseases.

Hypertrophy of the right ventricle.—The signs and symptoms of the *causes* of this enlargement will be found to be present. These causes may be briefly enumerated : (1) Obstruction in the pulmonary circuit, as emphysema and cirrhosis of the lung, bronchiectasis, and extensive obliteration of the pleural sac from adhesions ; (2) very rarely pulmonary valve-disease or congenital narrowing ; (3) mitral obstruction and regurgitation which interfere with the return of the blood from the lungs. The most important of these causes is mitral narrowing, in the compensation of which the left ventricle can take no part till too late—*i.e.* till after the failure of the compensation of the right heart.

Physical Signs.—There will be no symptoms, when the cause is mitral stenosis, till failure of compensation begins to show itself. If the patient be examined during the period of perfect adjustment, some bulging of the epigastrium and of the lower left costal cartilages—the fifth, sixth, and seventh —and of the lower part of the sternum may be discovered on inspection. The apex-beat is displaced outwards, but rarely beyond the nipple-line, and only slightly downwards ; it is very diffuse, and may generally be seen and felt superficially, as if directly under the hand, behind the sixth and seventh cartilages and the lower end of the sternum. The thrill occurring during auricular systole and terminating in the apex-beat, can usually be felt on placing the hand over the præcordium when mitral stenosis is present. The cardiac dulness is increased, extending usually in the transverse direction from the apex—viz. inside the nipple-line—to a point on the right side, which may be two inches or even more from the middle line of the sternum, where it joins the liver-dulness.

Auscultation reveals a clear, sharp, superficial first, and often a reduplicated second sound ; when stenosis is present, the characteristic auriculo-systolic murmur precedes the first sound. When at a later stage tricuspid regurgitation has supervened, visible pulsation in the jugular veins becomes apparent. Visible pulsation is usually marked in the epigastrium from an earlier period : it is caused by the contact of the hypertrophied ventricle with the liver ; but appearing at a later stage it may be hepatic pulsation due to venous regurgitation.

The signs and symptoms of hypertrophy of the right ventricle, caused by pulmonary obstruction, rarely remain entirely latent as in mitral stenosis, for the lung-symptoms obtrude themselves at an earlier date and attract attention to the cardiac condition.

Hypertrophy of the auricles is never simple ; there is invariably a very great degree of dilatation in all cases. The left auricle increases markedly in capacity, and its walls very considerably increase in thickness in mitral stenosis and in regurgitation. Owing to the situation of this chamber, its enlargement can only be inferred from the presence of obstructive or regurgitant mitral lesions, though sometimes the enlarged auricle may cause dulness in the left third intercostal space, with an auriculo-systolic pulsation visible in the second intercostal depression.

Right auricular hypertrophy with dilatation results from the same causes enumerated under ' Hypertrophy of the Right Ventricle,' with which it is always associated. The enlarged auricle will cause dulness extending beyond the margin of the sternum in the right third and fourth intercostal spaces, and occasionally an auriculo-systolic pulsation or wave may be seen in these spaces. Systolic pulsation, the result of tricuspid regurgitation, appears when the right ventricle has become dilated.

The tendency of dilatation ultimately to supervene in all cases of hypertrophy has been already insisted upon ; the determining cause may be some severe strain, an illness like influenza or one of the fevers, or the supervention of renal disease or the onset of old age.

Certain changes in distant parts may be brought about by the presence of the hypertrophy, though this is to be regarded as a conservative process, and an invariably beneficial one when considered with reference to the primary lesion. When the left ventricle becomes hypertrophied from peripheral resistance in the capillaries caused by the circulation of blood containing some irritating substance, the increased force of the pump may rupture an ill-supported vessel in the cerebral tissue or a miliary aneurism, and a subsequent apoplexy may result.

Another serious event may be traced to the powerful contraction of the hypertrophied ventricle in such a condition—sclerosis of the aorta and main arteries may be produced by the continuous high pressure, so that, though often the ventricular hypertrophy is the direct *result* of arterio-sclerosis, it may in some cases be the *cause* of it. Pulmonary apoplexy may likewise be the result of the hypertrophy of the right ventricle.

Diagnosis.—When the cardinal signs of hypertrophy are present there is little difficulty ; these are, as already mentioned, an apex-beat of exaggerated power and of a slow heaving character, displaced outwards and downwards, with markedly increased area of cardiac dulness.

Emphysema of the left lung may obscure these signs ; but the characters of the breath-sounds, taken together with the

strong high-tension pulse and the discovery of the original cause of the hypertrophy—*i.e.* valvular or renal disease—will prevent mistakes.

The opposite condition may be met with, in which, owing to the cicatrisation of a large cavity in the left lung, or owing to pulmonary contraction on the same side by pleuritic adhesions, a large portion of the healthy heart may come into direct contact with the chest-wall and simulate hypertrophy. In these cases there is some displacement of the heart towards the pulmonary apex ; hence the cardiac apex will be raised and the physical signs of the pulmonary disease will be present to reveal the true nature of the condition. The absence of the heaving character of the cardiac apex-beat will also help to prevent mistakes.

Pericardial effusion, with its pear-shaped area of dulness and the absence of the heaving impulse, with the characters of the pulse and the history of the case, is easily differentiated.

Pleuritic effusion and *aneurism* can only cause difficulty in the diagnosis by displacing the apex-beat, and a careful mapping out of the dulness will demonstrate their nature.

The increased pulsation seen in neurotic patients, especially when it occurs in young subjects with thin chest-walls, is not associated with displacement of the apex-beat, and is devoid of the characteristic slow heaving action of hypertrophied ventricles.

Dilatation, with its wavy, diffuse, irregular, and weak impulse, and the other signs of failure of the pumping power of the heart, is usually differentiated without much difficulty.

The diagnosis of right-sided from left-sided hypertrophy is already detailed upon pages 578 and 579.

Prognosis.—This will be the prognosis of the original disease inducing the hypertrophy ; most frequently it will be, therefore, that of a valvular disease or of cirrhotic kidney. When compensation has been thoroughly established in valvular disease the patient may live to old age ; this is seen in the comparatively rare cases of pure aortic obstruction which are in marked contrast, from a prognostic point of view, with aortic regurgitation—a disease in which death may occur in spite of enormous hypertrophy.

Failure of compensation is always serious ; but where its causes are removable, as muscular over-work, a short or transient feverish attack, or severe mental or emotional strain, there are often excellent grounds for hoping that restoration of the balance may be effected. In the writer's experience the prognosis is almost always grave when the disturbing element has been influenza. The hypertrophy of the heart seen in athletes, in Graves' disease, and in individuals following laborious occupations, is harmless for the most part, and when the causes are removed it may entirely disappear. Hyper-

trophy in Bright's disease is obviously grave, both from the point of view of the fatal nature of the kidney-affection and the liability to cerebral hæmorrhage.

The supervention of anginiform attacks in hypertrophy of the left ventricle indicates the advent of coronary disease, and they are the heralds of sudden death, which is usually not far off.

Treatment.—Hypertrophy of the heart requires no treatment, save in the few cases in which the cause is removable; when the physician discovers that this is possible, his duty is obvious. The aim should be to prevent failure of compensation by attention to diet, habits, rest, exercise, mental and physical labour, and everything which tends to raise and maintain the patient in a position of health and vigour. The different agents indicated in failing compensation will be mentioned when valvular lesions are described.

HEART, INFLAMMATIONS OF

(See under ENDOCARDITIS and PERICARDITIS. Myocarditis is described under 'Degenerations of the Heart,' on page 552.)

HEART, MALFORMATIONS OF

The acquired conditions of deformity of the valves are of much greater importance than the congenital varieties, most of which are only of pathological interest. Congenital malformations are, however, often the result of a fœtal endocarditis as well as the result of some perversion in the normal process of development. In some cases, moreover, the deformities resulting from developmental defects seem to induce an endocarditis during fœtal life which still further intensifies or exaggerates the primary malformation. The subject is still further complicated by the fact that after birth the malformed valves and septa may become the seat of endocardial inflammation in those cases where the infant survives. Cautley regards syphilis as a common cause of congenital heart-disease.

The list of congenital malformations is a long one, and usually several occur in the same subject; they are often found in association with other developmental defects, as hare-lip, talipes, spina bifida, &c., but since some of them are incompatible with extra-uterine life they are of no clinical interest.

Patent foramen ovale is one of the most common of the congenital cardiac malformations; it may exist alone, but it often is associated with stenosis of the pulmonary artery and

with defects of the ventricular septum. In this condition the blood flows through the opening in the septum between the auricles from the right directly to the left, instead of passing into the right ventricle on its route to the lungs. The defect is caused by failure in the development of the membrane of the fossa ovalis after birth ; usually the failure is only partial, a slit being left which remains during life without producing either symptoms or signs of cardiac imperfection.

Many cases are recorded where the aperture was of considerable size, and the condition was only discovered after death from some other affection late in life, no symptoms having been observed ; but in other cases the clinical picture of congenital heart-disease has been observed.

Absence of the auricular septum may be complete, producing a heart with one auricle and two ventricles ; if the interventricular partition may be missing at the same time the heart consists of but two cavities—an auricle and a ventricle, out of which a vessel springs to supply the lungs and carry on the systemic circulation, as is the normal condition of the reptilian heart.

When the ventricular septum is completely absent the result is a heart with one ventricle and two auricles, though both the mitral and tricuspid openings may be present, and the single vessel divides into an aorta and pulmonary artery. More frequently the absence of the ventricular septum is only partial, and the deficiency is usually located in the region of the pars membranacea or the so-called ' undefended space,' causing an aperture through which both ventricles are in communication with each other. Life may be prolonged past the middle period under such circumstances if hypertrophy meets the increased demand on the circulatory mechanism.

Stenosis of the pulmonary artery or of the right conus arteriosus is even more common than patency of the foramen ovale, being present in the great majority of congenital heart-cases which survive the period of childhood ; it is usually associated with openings in the ventricular and auricular septa, and with patency of the ductus arteriosus. Atresia of the pulmonary artery is much less common and more serious. Both conditions are caused by fœtal inflammatory mischief affecting the valves or involving the vessel above their origin ; it may, however, be produced by a developmental defect in the truncus arteriosus. Pulmonary tuberculosis and cerebral disease are liable to supervene in those cases which survive the period of childhood.

Stenosis and atresia of the aorta are comparatively rare. When the narrowing occurs at the orifice of the ductus arteriosus in the beginning of the descending aorta the condition

may give rise to no symptoms till the period of middle-life is reached.

Patent ductus arteriosus sometimes exists without the cardiac malformation, and there may be no symptoms till the compensatory hypertrophy of the left ventricle breaks down at a late stage.

Transposition of the aorta and pulmonary trunks may exist without signs or symptoms, but usually the subjects of this malformation do not survive infancy.

Valvular deformities the result of fœtal endocarditis of the sclerotic type and of defects in development are often met with in the semilunar valves, but rarely in the mitral or tricuspid; all four sets of valves may also be implicated in the same subject. The malformed valves may afterwards become the site of inflammatory changes and further deformities as life advances. The malformation may arise from an increase in the number of the semilunar segments, or from a bicuspid arrangement, or from complete fusion of the segments. When the auriculo-ventricular valves are affected the segments are united or fused together as seen in valvular disease following rheumatism. Whilst the acquired valvular disease secondary to rheumatic endocarditis in post-fœtal life is almost always confined to the left side of the heart, in the congenital cases right-sided valve-lesions are comparatively common.

Premature closure of the ductus arteriosus, which should not become obliterated till after the seventh or fourteenth day following birth, is sometimes, though rarely, met with. Premature closure of the foramen ovale is one of the rarest malformations, though at birth little blood passes through it.

Displacements of the heart are met with in which the organ may be found high up in the neck, or below the diaphragm, or protruding in a parietal thoracic fissure covered over only by membrane and integument—*ectopia cordis* or *ectocardia*.

Transposition of the heart (*dextrocardia*) is nearly always associated with other visceral misplacements, but the organ may be found occupying the centre of the thorax without displacement of other organs, in which cases it is always the seat of malformations. So-called 'double heart,' or dugong heart, is produced by a cleavage at the apex which is an exaggeration of the ventricular sulcus.

Absence of heart, or *acardia*, is sometimes met with in still-born monsters, and absence of the pericardium more or less complete has been many times recorded.

Symptoms.—As already stated, many varieties of cardiac deformity may exist without the production of any symptoms, whilst some may only give rise to symptoms at a late period in life.

The chief symptom or sign of congenital heart-disease is *cyanosis*, which is present in the great majority of all cases, so that the term *morbus cæruleus* is often applied to the group of malformations of the heart of congenital origin.

The blue tint varies according to the severity of the case; sometimes it is a dark purple surpassing in degree any discoloration to be met with in disease save in the last hours of life in some asphyxial conditions. The tint is deeper on the lips, nose, ears, and finger-tips, but in grave cases the entire cutaneous surface and visible mucous membranes show it, and it is increased on exertion. It usually appears immediately after birth, but in rare cases it has not become obvious till the child has survived several years.

In slight cases the discoloration only becomes marked when the patient is chilled or out of sorts, or has taken exercise, but in all cases it is liable to change in depth from day to day.

Slight œdema is sometimes present—*i.e.* more or less puffiness in those parts which, as just stated, are the site of the deepest purple colour. The skin and the extremities feel cold, though the rectal temperature may not be reduced. Respiration is quickened and dyspnœa is easily induced on exertion, which may pass into convulsions or attacks of prolonged unconsciousness in infants and young children.

Clubbing of the fingers and toes is always a marked feature when the cyanosis is severe; it is caused by an enlargement of the terminal phalanges, and is indicative of severe venous engorgement; the nails are incurved. The mental condition is sluggish; the child is incapable of exertion and does not thrive; it often presents a wasted appearance, or looks as if the general development had to some extent become arrested. At a later stage general œdema, ascites, constant cough and sometimes hæmoptysis, supervene; and many patients succumb to pulmonary tuberculosis.

The blood shows remarkable characters; it is concentrated, and the count reveals a large increase in the red discs, which may number up to nine millions in a cubic millimetre; the hæmoglobin may rise to 160 per cent., and the s.g. to 1080.

The old hypothesis that the cyanosis was the direct result of the mixture of arterial and venous blood must now be abandoned, since numerous cases are to be met with where this blending takes place without any lividity, as in examples of the disease where only one ventricle is present, and in others where the arterial and venous blood freely commingle without any resulting cyanosis.

Morgagni maintained that the lividity was the result of the general venous congestion caused by the mechanical obstruction to the circulation.

The view of Lees and Morison is more probably correct—that the lividity is the result of imperfect aëration of the blood in the lungs owing to obstruction of the pulmonary artery.

The physical signs in the cardiac region are varied, and though they serve to corroborate the diagnosis of congenital heart-disease they often fail to demonstrate the peculiar malformation present. There are usually evidences of hypertrophy and dilatation, as observable in some bulging with an apex-beat displaced outwards and visible over an extended area. Cardiac dulness is much increased in extent, and on auscultation a loud systolic murmur is heard all over the precordial region, often without any localised spot where its maximum intensity is obvious, and not propagated in any particular line of direction.

In pulmonary stenosis, which is the most important and most frequently present congenital malformation, there is much increase in the area of cardiac dulness beyond the right margin of the sternum, owing to the hypertrophy of the right ventricle ; the apex may be visible and palpable below the xiphoid, and a systolic thrill can be felt, especially towards the upper limit of cardiac dulness—*i.e.* in the second and third spaces on the left side. A loud systolic murmur is usually present, heard all over the præcordium and most intense in the region where the thrill is felt. This blowing murmur is conducted along the vessels in the neck. The second sound in the pulmonary area is usually absent or feeble ; but occasionally it is loud and ringing, in which case it is probably caused by a patent ductus arteriosus. The abnormal weakness of the pulmonary second sound in association with a systolic murmur is regarded by Hochsinger as diagnostic.

In patency of the foramen ovale, as already mentioned, symptoms and signs may be entirely absent. Sometimes there is marked cyanosis without murmurs ; but even when cyanosis is absent, presystolic, systolic, and diastolic murmurs may be heard, which, however, only tend to obscure the diagnosis.

Patency of the ductus arteriosus, like the condition just mentioned, cannot usually be diagnosed during life, and the same remark applies to transposition of the great vessels, stenosis of the aorta, and to imperfections in the ventricular septum.

Diagnosis.—The question of the congenital or acquired origin of a given cardiac murmur in a young child may be in some instances a difficult one. When marked cyanosis exists from an early period after birth, there can be little doubt of the congenital nature of the case, because the characteristic deep lividity and clubbing of the fingers never follow the ordinary post-natal form of valvular disease.

In the absence of marked lividity the difficulty may be great, but the congenital murmurs, which are usually systolic in time, and loud, are not typical like the acquired murmurs as regards their site of maximum intensity and direction of conduction. It should be remembered also that a considerable degree of hypertrophy of the heart requires a fair amount of time for its development, so that a loud murmur without a markedly increased area of cardiac dulness in a very young subject would be in favour of congenital origin, since a considerable degree of hypertrophic enlargement is the exception in this condition.

Where the hypertrophy in young children is obviously confined to the right ventricle, as demonstrated by the cardiac dulness and absence of increased force in the apex-beat, the diagnosis of congenital disease will probably be correct. Recently Ballantyne and McCrae have drawn attention to the fact that congenital heart-disease may be diagnosed during fœtal life by careful auscultation over the uterine region during the later months of pregnancy.

Prognosis.—Many of the congenital malformations, when severe in extent, are incompatible with lengthened life, as seen in those in which there is complete absence of the ventricular septum and atresia of the pulmonary artery and aorta, the subjects of which usually survive only for a brief period after birth. Where cyanosis and clubbing of the fingers are very marked at an early period, and where the heart continues to enlarge, the duration of life is almost certain to be brief. Death in any case may be sudden.

In pulmonary stenosis the chance of the patient surviving to middle-life largely depends upon the presence of apertures in the septa which relieve the overloaded right heart; and the presence of these are rendered probable by murmurs heard over the base, especially if there be not an apical murmur and thrill.

In vigorous children with slight cyanosis and little clubbing life may be prolonged to the usual average, but the supervention of bronchitis and phthisis is always an element to be remembered. Poynton states that the majority of all subjects exhibiting congenital heart-disease die before the end of the second year after birth.

Treatment.—Little can be done save the paying of close attention to every hygienic law which tends to improve vigour and protect the patient from bronchial attacks. Diet, clothing, climate, rest, and exercise require careful consideration. Complications must be treated promptly on the lines laid down for the management of chronic valvular affections, the most important remedy being prolonged rest and the discreet administration of cardiac tonics.

HEART, THROMBOSIS OF.

(See under THROMBOSIS.)

HEART, VALVULAR DISEASES OF

Etiology and Pathology.—The most common cause of chronic disease of the valves is the form of endocarditis which occurs as a result of the infective process met with in acute rheumatism. Under 'Endocarditis,' on page 373, the resulting valvulitis which chiefly affects the left side of the heart and its pathological anatomy are described. The main element in the diseased process which eventuates in chronic valvular affection, as already detailed, is the sclerotic change which results from the formation of a fibrous cicatricial tissue, which, as life advances, tends to contract. This contraction produces shortening, thickening, and sometimes fusion or gluing together of the valve-segments, whereby the function of the valve is seriously interfered with, so that obstruction to the flow of blood results, or the opposite condition is induced in which the deformed valve-segments fail to approximate, permitting regurgitation to take place; or both results may be produced at the same valve. With these changes there is a tendency towards the deposition of calcareous salts and recurrent attacks of valvulitis, which produce still further deformity.

The chordæ tendineæ also take part in the morbid process, which first involves their valvular extremities and gradually extends towards their muscular attachments, causing great shortening. In advanced cases fibroid changes are observable in the elements of the musculi papillares at their tendinous extremities, and sometimes the chordæ tendineæ coalesce.

As stated under 'Endocarditis,' the original endocardial mischief is not confined to the specific action of the rheumatic micro-organism, but may be seen to follow various other conditions, as typhoid fever, scarlatina, Bright's disease, gonorrhœa, erysipelas, gout, syphilis, &c. Other causes of chronic valvular affections are prolonged muscular strain and trauma, which may induce sclerotic changes of a degenerative type in the valves; these do not strictly fall under the designation of an inflammatory process. Atheroma belongs to this group, and is not infrequently met with as a cause of aortic valve-disease producing regurgitation, though here it is but a local manifestation of a widespread arterial degeneration, often associated with syphilis. A most important group of etiological factors in producing regurgitation at the mitral and tricuspid valves are those lesions of a degenerative type which induce dilatation of the ventricles, and so cause a *relative* insufficiency

—an exaggeration of the normal condition in the tricuspid. These affections, with their murmurs, are not illustrations of valve-disease, but are to be considered as the results of dilatation from whatever cause.

Heredity plays but a small part in valvular disease, but instances are on record which tend to show that in some families an hereditary vulnerability of the heart to rheumatism may be a factor.

Age has its chief influence as a determining factor in the incidence of acute rheumatism, which is most common in young adults, but the symptoms of valve-disease often do not show themselves for many years after. No age is exempt, and valvular disease is not very rare even in fœtal life. Aortic lesions are more commonly met with at middle-life or later.

Sex.—Upon the whole, men are the subjects of valve-disease more frequently than women, but mitral stenosis is much more common in women and aortic disease in men.

Occupation shows its influence by the greater frequency of the disease, especially aortic regurgitation, in those whose daily work entails severe muscular labour.

Mitral disease is the most frequently met with form of valvular affection, representing more than half of all the cases of valvular disease, and regurgitation is much more common than stenosis at this orifice—indeed, pure mitral stenosis is comparatively infrequent; but a combination of stenosis and regurgitation, though perhaps not so common as simple regurgitation, is very frequently met with.

Aortic regurgitation, alone or combined with stenosis, represents about one-fifth of all valvular cases, but pure aortic stenosis is one of the rarest of diseases. A common combination is mitral regurgitation and aortic regurgitation, but the former may be a direct sequel or result of the latter.

Pulmonary valve-disease is rare, though murmurs are common in this area. The affection when met with is likely to be of congenital origin; the same may be said of tricuspid regurgitation when existing alone; tricuspid regurgitation, however, is a very common result of mitral disease owing to relative insufficiency when there is no disease of the tricuspid valve.

The effects of valvular disease on the heart-walls and upon other organs is a subject of the greatest importance, for without a knowledge of the altered dynamics of the damaged organ it is impossible to interpret the varied symptoms which arise during the progress of valvular disease. The subject is simplified by a study of the means by which nature attempts to repair the injury to the circulation caused by the valvular defect. Little is to be hoped in the direction of any improvement in the condition of the damaged valve, because as a rule, owing to the progressive nature of the pathological changes

in the cicatricial fibrous tissue, the tendency is towards further shortening or thickening of the already deformed segments.

The natural effort at repair proceeds in the direction of establishing compensatory changes in the muscular walls of the cardiac chambers.

Compensation.—When narrowing or stenosis occurs at any cardiac orifice the rule is that the muscular wall of the chamber behind the obstruction hypertrophies. Fortunately the progress of the narrowing is an essentially slow one, and permits of time for an increase in the size and number of the muscular elements, so that the natural effort is often entirely successful in overcoming the obstruction without the production of any symptoms of embarrassment of the heart's action or interference with the general circulation. When this condition is arrived at, the stage of perfect compensation is said to be reached. This is witnessed in its most complete form in the rare condition of pure stenosis of the aortic valves, where the left ventricle enormously increases in weight and strength. In a somewhat less typical degree it is observed in mitral stenosis where the left auricle takes on hypertrophy.

In aortic regurgitation, after the closure of the insufficient valves a portion of the blood regurgitates into the left ventricle ; another element is introduced then into the natural effort to effect compensation or the restoration of the balance. It is obvious that a mere hypertrophy of the muscular wall could not of itself achieve this end ; hence an increase in its capacity must be provided for, so that at each stroke of the ventricular pump an additional quantity of blood may be driven into the aorta to make up for the amount which regurgitates during each diastole. In order to meet this requirement, the ventricle dilates owing to its continual over-distension, and at the same time its walls increase in thickness and strength. In this way compensation is effected by the production of the condition known as eccentric hypertrophy, or hypertrophy with dilatation.

Often, however, the change does not stop here ; should the regurgitation be extensive, the necessary degree of dilatation of the ventricle may be so great as to cause yielding or widening of the ring supporting the mitral valve, which will produce regurgitation into the left auricle. (This is the condition known as *relative* insufficiency.)

To pursue the changes induced by this new condition of affairs, the effects which the regurgitant column of blood produces in the left auricle must be considered. This cavity becomes continually over-distended and its blood-pressure elevated, and to overcome its increased burden dilatation and hypertrophy of its walls must ensue. The obstruction to the flow of blood returning from the lungs, through the pulmonary veins discharging into the distended auricle, tends

towards passive congestion of the lungs, to overcome which the right ventricle takes on compensatory hypertrophy. By these successive steps compensation is at length effected, and the leakage at the aortic orifice remedied by calling into action the reserve force of the right side of the heart.

If, however, instead of beginning with a study of the changes induced by aortic regurgitation, the starting-point of mitral stenosis be selected, similar sequences of events may be observed: the left auricle behind the narrowed mitral valve dilates and hypertrophies, and ultimately, as the pressure in the pulmonary veins increases, the right ventricle by its hypertrophy restores the balance. Sometimes the changes proceed still further, and as the right ventricle dilates to a great extent the tricuspid valve yields (as the mitral does in aortic regurgitation), and regurgitation takes place into the veins, causing pulsation in the jugulars and sometimes in the liver, unless the right auricle dilates and hypertrophies to an extent sufficient to prevent this. Finally, if mitral regurgitation be considered, identical processes are found at work. The left auricle becomes distended by the regurgitant column of blood; and as it must receive this, in addition to the normal amount returning from the lungs through the pulmonary veins, a large degree of dilatation and hypertrophy must ensue in order to propel into the ventricle an increased quantity of blood so as to allow for the amount which comes back to it through regurgitation. This additional charge causes dilatation and hypertrophy of the left ventricle. Whilst this change is taking place in front of the auricle, equally important results are occurring behind it. Owing to the increased pressure in the auricular chamber, the pulmonary circulation is carried on under difficulty, to overcome which the right ventricle dilates and hypertrophies, and usually the increased pressure in it leads to hypertrophy and dilatation of the right auricle. Under any of the above conditions the equilibrium may be so perfectly adjusted that the subject of valvular disease may live for many years without even a suspicion that anything is amiss with his heart, unless some unusually great demand be made upon what is called

The Reserve Energy of the Heart.—Under ordinary circumstances the full power of the heart in health is not brought into action, but in sudden spurts of intense muscular activity the right chambers become distended and the utmost reserve power of both ventricles and auricles is rapidly called into action to overcome the temporary embarrassment to the circulation. In valvular disease compensation can never be strictly said to be *perfect*. The increased demand made upon the damaged organ in carrying on the circulation uses up a considerable amount of this reserve force, but the natural process of compensation by

hypertrophy to a certain degree remedies this by increasing the total potential energy of the heart. There is thus in the most completely compensated cases of valve-disease a condition in which the power of the heart is increased above that of the normal; but as most of this power is required to keep the organ working in ordinary states of rest and tranquillity, there is therefore only a slight degree of energy left in reserve.

Hence it is that, though the compensated heart may be working perfectly for years without embarrassment of any kind, should a very great demand be suddenly made upon it—such a demand as could be met in the healthy heart by its great reserve power—an inevitable breakdown occurs. This demand may be made in the form of violent muscular exercise as in running or hill-climbing, or it may be a pneumonia or acute bronchitis which suddenly calls for more exertion from the right ventricle, or it may be a prolonged series of exertions which necessitate the hypertrophied organ to keep working up to its utmost limit of power.

Failure of Compensation. — Under the conditions of muscular strain just mentioned, the hypertrophied heart being unable to overcome the increased mechanical resistance caused by the valvular imperfection, the stage of failing compensation is reached. This state is also produced by disease and by every condition which, interfering with normal nutrition, causes deterioration of the cardiac muscular fibre, as improper or deficient food, prolonged anxiety or mental strain, advancing age, degenerative changes, &c. Of the diseases which induce failure of compensation, none has been so frequently met with in recent years as influenza; but all the members of the group of fevers are answerable for cases of cardiac breakdown, owing to the injurious effects of their toxins upon the cardiac muscle. Much will depend upon the period of life at which the valvular lesion is established. In young robust males the onset of obstruction is met by a generous amount of hypertrophy, which, in the face of the increased demands made upon the power of the heart by the activity of youthful exercises, reaches a high degree of compensation. The degree or amount of natural repair may be so great in such cases as to lead to the creation of a reserve power almost, but perhaps never as high as in health. In these cases active or even athletic exercises are indulged in without causing any disturbance of compensation. (The case is, however, different in children: though the valve-deformity may even remain stationary, the necessary growth of the heart as the body increases in bulk intensifies the damage relatively in stenotic lesions.)

When, upon the other hand, the valve-mischief makes its first appearance in middle or later life, the natural efforts at compensation may only succeed in producing an amount of

hypertrophy which just enables the circulation to be carried on effectually when the patient is in a state of physical rest, the entire reserve force of the heart being continually in action to meet the demands made upon the circulation by the valvular impediment. In such cases the slightest exertion brings on some of the symptoms of failing compensation presently to be described.

By way of contrast, an interesting and instructive demonstration of nature's method of effecting compensation in chronic valvular disease is obtained by a study of those rare cases of trauma where, owing to a violent muscular effort, a segment of the aortic valve is ruptured. Here, there being no time for the slow process of hypertrophy to occur, sudden death or the rapid supervention of dilatation, and the other signs and symptoms identical with those met with in failure of compensation, appear.

Symptoms of Failing Compensation.—The recognition of these is a matter of the utmost importance, as they very generally are the causes which first induce the subject of valvular disease to seek advice.

Breathlessness on exertion is usually the first symptom to attract attention. It may be so slight that the patient fails to notice it, and the physician is often informed that there is no breathlessness, when the trained eye will detect it upon the slight exertion necessitated by a physical examination. It soon amounts to dyspnœa following such exercise as rapidly ascending even a short flight of steps, and its absence is almost a sure indication of the efficiency of compensation. Often attacks of breathlessness occur at night, waking the patient from sleep; they are caused probably by some increase of blood-pressure, brought about by postural influences.

Palpitation is nearly always associated with the dyspnœa or breathlessness, and with it various degrees of precordial discomfort are experienced.

Cough is likely to be overlooked, as the patient becomes so accustomed to it as to be unconscious of its presence; it is especially liable to be induced by exertion, and at a later stage the other symptoms and signs of bronchial catarrh and œdema supervene, even to the extent of profuse hæmoptysis when the lungs become seriously congested. Finally brown induration of the lungs is induced by the chronic congestion; it is often preceded by the kind of solidification known as red induration, which renders the lower lobes airless and œdematous. Pulmonary infarction is also common.

Digestive disturbances are common, owing to hepatic stasis.

Congestion of the liver may be an early sign, and the tender and enlarged organ may be palpated below the costal arch. The swelling of the liver is caused by the stagnation of the blood in the right auricle, which retards the discharge of the

blood from the hepatic vein into the distended vena cava. The amount of enlargement varies widely with the state of venous stasis from day to day. When of long duration, *jaundice*, *ascites*, and the form of hepatic change known as the *nutmeg liver* result, and finally atrophy of the organ, may supervene.

Dropsy, or anasarca, is a very important early sign of failure of compensation. As a rule it is not perceived by the patient in this very early stage, being manifest only by slight pitting over the front of the tibia; even in severe and prolonged cases it is usually confined to the lower extremities, but towards the final stages it may become general even when the kidneys are sound.

Dropsy of the pleura or hydrothorax, is common, and generally bilateral; some pericardial dropsy is usually present.

Splenic enlargement and *renal congestion*, with albuminuria, are present at a late stage.

With these symptoms and signs are always found evidences of dilatation of the heart.

Irregularity of the pulse.—This is an important clinical phenomenon, upon which much light has been recently thrown by the researches of Mackenzie, Gaskell, John Hay, Erlanger, Hering, Keith, and others. The irregularity or arrhythmia is of the type of the *disorderly rhythm* described by Mackenzie as due to abnormal inception of the cardiac rhythm, the ventricles and auricles contracting simultaneously, owing to disturbances arising in the auriculo-ventricular bundle of fibres of His. The subject is of great importance, owing to its relationship to the causation of heart-block referred to upon page 568. The recent researches of Mackenzie are summarised under 'Arrhythmia,' upon page 571.

The cardiac embarrassment may be sometimes accompanied by pain, often as severe as that met with in typical angina pectoris, and it is probably also caused by secondary coronary disease.

MURMURS.—The valvular lesion is always accompanied by the characteristic bruit or murmur which, to prevent repetition, will be described under the heading of each form of the disease. This is an adventitious sound produced within the heart at one of its orifices; hence it is designated endocardial in contradistinction to those exocardial sounds which originate outside the organ.

The production of these endocardial murmurs is usually held to be the result of eddies, or of what is known as a *fluid vein*. The blood, rushing through a narrow opening into a wide chamber or channel beyond it, has its particles in a state of vibration, which is conveyed to the walls of the chamber or vessel against which the jet impinges. It also seems certain that, without any appreciable narrowing, a murmur may be

produced by the vibrations of small pedunculated growths on the fringe of a diseased valve; these certainly intensify a murmur when the conditions which produce the fluid vein are also present. Thinness of the blood probably is no factor, as formerly supposed, since endocardial murmurs of great intensity are heard in congenital valvular malformations in which the blood is in a remarkably concentrated state. A requisite degree of blood-pressure seems to be necessary, since increased cardiac force intensifies most murmurs, and the opposite condition weakens them or renders them inaudible.

The murmur is always conveyed in the direction of the blood-stream in which it originates; thus in stenosis of the aorta it is heard along the great vessels, whilst in regurgitation at the same orifice the sound is conducted by the regurgitated jet of blood downwards along the sternum. In mitral regurgitation the murmur is audible behind, being conveyed backwards by the jet of blood driven into the deep-seated enlarged auricle.

The murmur may accompany, precede, or take the place of a normal cardiac sound.

The following points should be noted in the investigation of an endocardial murmur:

Quality or timbre.—Usually this feature is of minor consideration; but in the recognition of one murmur (*i.e.* that of mitral obstruction) it is a matter of much importance. Often the adventitious sound has an unmistakable 'blowing' quality or character, and if attention be concentrated upon such a sound these points are obvious: the murmur begins faintly or softly, but it reaches its maximum very soon after its commencement and gradually fades away into silence, so that the ear often fails to determine the precise moment at which the sound stops. In marked contrast to this blowing sound is the crescendo murmur, indicative of mitral stenosis, which, commencing almost inaudibly, rapidly increases in intensity and stops abruptly on the advent of the first sound.

Harshness, softness, musical qualities, intensity, churning, rasping or grating characters, &c., and many other modifications of sound have no diagnostic signification, and, like mere loudness, give no indication whatever of the extent or character of the valve-lesion. But marked loudness or intensity often affords an indication of the power of the ventricle or auricle, since it is rare to find a very loud murmur when muscular power is failing. Stenotic murmurs are generally harsher in their tone than regurgitant ones. Some idea of the extent of the lesion may be obtained by noting whether or not any of the normal heart-sound accompanies the murmur, because, if the latter *entirely* replaces the true or normal valvular sound, it is probable that the function of the valve is very seriously in abeyance; this point is of considerable importance in aortic regurgitation.

Site of maximum intensity.—The spot on the chest-wall at which the murmur is heard loudest should be noticed; this is not necessarily immediately over the area at which the normal sound is most intense. Mitral regurgitant murmurs have their point of maximum intensity in the apex-region or immediately outside it, whilst stenotic murmurs produced at the same orifice are loudest just inside the apex-area.

Direction in which the murmur is conducted.—As already stated, the adventitious sound is conducted in the direction of the *veine fluide* which produces it, and the bruits of mitral and aortic regurgitation are propagated along definite lines of direction on the chest-walls. Thus the mitral murmur is conveyed from the apex outwards towards the axilla, and may be heard in some cases in thin subjects without a break all along a horizontal line extending from the apex to the spine.

Often, however, an endocardial murmur may be so intense as to be audible over the entire thorax and even beyond it; this is most frequently the case with rough, loud aortic regurgitant murmurs, the result of primary sclerotic changes.

Rhythm.—Though mentioned last, this is the main problem to be solved in the diagnosis of valvular diseases. The rhythm or 'time' of a murmur is the relation which it bears to the systole or diastole of the ventricles. Speaking roughly, its rhythm or time may be also said to be its relation to the first or second sound. In the largest number of cases met with, the problem of rhythm is simply the relation between the murmur and the apex-beat, since this latter is synchronous with the ventricular systole, first sound of the heart and carotid pulse. Murmurs are said to be *systolic* when they begin with the first sound—*i.e.* when they are produced during the ventricular systole. *Diastolic* murmurs are produced during ventricular diastole, and accompany the second sound, or are audible between it and the succeeding first sound. The *presystolic* murmur occurs immediately before the first sound, and is produced at the moment of auricular systole; it is therefore diastolic, and its proper nomenclature, as insisted upon by Sir W. Gairdner, is the *auriculo-systolic* murmur. (The varieties known as middle and late diastolic will be considered under 'Mitral Stenosis.')

The diagnosis of the rhythm of an endocardial murmur is as a rule free from difficulty *when the normal first or second sound, which it accompanies, remains distinctly audible.* Unfortunately, however, in a considerable percentage of cases the murmur takes the place of the normal sound, and the difficulty may be further increased by the fact that the remaining normal sound and the relative duration of the pauses are modified by cardiac arrhythmia. The usual procedure in such cases is to time the murmur by noting its relation to the apex-beat. If it occurs at the moment when the

impulse is felt against the chest-wall, it must obviously be systolic. The difficulty to the student and young practitioner in satisfying himself about the synchronism of these two events is a real one, as the writer knows by an experience of a quarter of a century's clinical teaching. An analysis of the difficulty shows that it exists in the attempt to register and contrast simultaneously in the sensorium two impressions, one of which is conveyed by the nerves of touch, and the other by the sense of hearing—a feat which is impossible without prolonged and patient practice. In this statement it is assumed that the finger is placed over the apex-beat area, while the stethoscope is applied to the ear. If the sense of vision be substituted for that of touch, the problem is easier; and an attempt is upon the whole more likely to be successful if the apex-beat is watched by the eye, while the ear listens to the murmur. But again the difficulty of coördinating the impressions conveyed by two different senses—hearing and vision—also maintains.

That the confusion of perception is not a theoretical matter anyone can convince himself by a simple kindred experiment. In mitral stenosis with a well-marked auriculo-systolic thrill, if the hand be placed over the heart, the thrill and the apex-beat are both palpable, and there is no difficulty in timing either, because the same sense—that of touch—only is employed in the solution of the problem.

If the factors in the difficulty of timing a murmur in which there is no normal first sound could be reduced to their simplest elements, the problem would be made easy.

This can be accomplished sometimes by the use of the differential stethoscope—*i.e.* a binaural one, with two separate chest-pieces. One bell is placed over the apex and the murmur is heard in one ear, whilst the other chest-piece is placed over the tricuspid area and the corresponding ear hears the normal tricuspid part of the first sound, which may be perceived to synchronise or not with the adventitious sound.

By the following method the writer has still further simplified the problem for the student in cases where there is a sharp or punctiform apex-beat. A piece of cork, with a short flexible silver wire attached, is fastened over the apex-beat by adhesive plaster, the wire moving up and down or to and fro at each systole; the bell of the ordinary stethoscope is then placed close to the cork, and the wire so manipulated and bent that its tip will strike the chest-piece of the instrument at each stroke, making a gentle tap. If the ears be now applied to the instrument, the auditory nerves will convey to the sensorium two unmistakable sounds, one of which—that caused by the contact of the wire upon the chest-piece—may be taken as an artificial first sound, which can then be easily appreciated as synchronising or not with the murmur.

Where there is a murmur not associated with the normal sound, and where the apex-beat is also absent, the carotid pulse may be taken as synchronous with the missing first sound. The method of timing a murmur by feeling the radial pulse whilst the stethoscope is over the apex is beset with so many fallacies that, especially in a quickly acting heart, it should not be attempted. The radial pulse is later than the first sound, and the time occupied by the transit of the impression from the finger-tip to the sensorium, though exceedingly brief, is still a source of error in a problem where a minute fraction of a second is of vital importance.

In all cases where the presence of murmurs raises the suspicion of chronic valvular disease, the inevitable secondary changes in the heart must be carefully sought for and found before the murmur can be accepted as of organic origin.

Functional murmurs are very common in anæmia and chlorosis, and their causation in these and other allied states is a subject of much dispute. Usually attributed to the thinness of the blood, there is strong ground for believing that the main factor in their production is some alteration in the calibre of the vessels or some slight transient dilatation of the chambers of the heart. They occur most frequently in the pulmonary area, but are sometimes met with at the apex. They are systolic in time, soft and blowing, and generally associated with a venous hum in the neck-veins. In anæmic subjects the apex-beat is not displaced, as there is no evidence of cardiac hypertrophy, and a rheumatic history is usually wanting. An aortic systolic murmur often does not mean aortic obstruction. It may be produced when the valves are intact by a *veine fluide*, caused by the blood being driven into a dilated aorta beyond the valves, and it may be produced in anæmia ; but the difficulty is easily settled by noting the absence of hypertrophy, which is invariably present when organic stenosis of the valve-orifice exists. The same remarks apply to those cases where a slight deformity on the cusp of one of the semilunar valves, without diminishing the size of the orifice, is still capable of producing a murmur.

VALVULAR DISEASE—AORTIC OBSTRUCTION

This lesion, uncomplicated by regurgitation, is one of the rarest of diseases. When aortic stenosis or obstruction exists, there is almost always insufficiency, the deformity preventing the perfect closure of the segments during diastole. These are usually adherent and thickened, and in old cases infiltrated with lime-salts, so that but a mere chink or slit is provided for the blood-stream. In many cases the stenosis is the result of contraction in the fibrous ring, the mischief being confined to the attached bases of the segments, the cusps being unaffected. Dilatation of the left ventricle is said to be always

present ; but, if so, it is very slight in degree, since the hypertrophy seems so pure that the term 'concentric' is usually applied to it.

Etiology.—The causes are those already mentioned in speaking of valvular disease in general, and they will be more particularly detailed under 'Aortic Regurgitation.' The disease is one of late or past middle-life, and more common in males.

Symptoms.—The affection may be latent all through life, and in its symptomatology it contrasts markedly with aortic regurgitation, in which the evidences of disturbance of the circulation are often numerous. Pain in the cardiac region is not, however, rare after exertion, but more commonly a sense of oppression or constriction behind the middle of the sternum may be complained of. As the patient advances in age, if muscle-failure supervene, the hypertrophy gives way to dilatation, and the complex symptoms caused by regurgitation through the mitral valve may be observed. This is owing to *relative* insufficiency, the dilated ventricle failing to support the mitral curtains, owing to widening of its muscular ring, in which case the backward pressure will tell upon the left auricle, lungs, and ultimately upon the right heart, as described upon page 590.

Physical Signs.—*Inspection* may reveal, contrary to what might be expected, an apparently normal or absent apex-beat, because the condition is most frequently met with in elderly subjects with rigid chest-walls and more or less emphysema. According to some authorities, the apex-beat is diminished in all cases. This statement is incorrect : sometimes, especially in young subjects, the visible impulse is obviously increased, and it is usually displaced slightly downwards.

Palpation gives evidence of a slow, heaving, steady impulse ; in one case graphically described by Allbutt 'the heart was heaving under the hand as the back of some imprisoned monster.'

A thrill is felt when the hand is applied over the aortic area. This may be palpable all over the aortic region from the manubrium to its origin in the ventricle ; it is systolic in time, and though also felt in dilatation of the aorta, it is a sign of much value when very palpable.

Percussion shows usually an appreciable increase in the area of cardiac dulness, but never to the same extent as in aortic regurgitation. The transverse dulness may be normal, but in the vertical direction the increase is more or less marked, owing to the hypertrophy lengthening the cone-shape of the ventricle.

Auscultation reveals a rough, harsh, noisy murmur, whose point of maximum intensity is at the aortic area—viz. the second right costal cartilage near to the sternum, and whose

direction of propagation is upwards along the vessels in the neck; its rhythm is systolic. Occasionally the murmur may be so intense as to be heard all over the thorax and even at a distance from the body, and sometimes it is of musical quality.

The second sound in the aortic area is usually feeble and often absent, as the fusion of the segments does not admit of such apposition as would cause an aortic second sound. The pulse in aortic stenosis is small and slow: it may be of the *anacrotic* type, which shows a sloping upstroke whose apex is formed by the tidal wave, and the dicrotic wave on the line of descent is but slightly marked. The *bisferiens* pulse is also met with, but less frequently. Its sphygmogram is very characteristic; the upstroke is high and vertical, with an acute apex, and the tidal wave rises in a similar manner, so that a deep cleft is left between them, giving the appearance of a deeply bifid apex. This variety of pulse is detected by the finger as consisting of two pulsations, the second produced by the tidal wave being stronger than the first, which is caused by the sudden distension of the artery.

A rate of forty is not unusual, and if the pulse be felt by the finger of one hand, whilst the other hand is pressed firmly against the patient's chest, the marked contrast of the slowly heaving strong ventricular contraction to the *pulsus parvus* is very striking.

Diagnosis.—As stated upon page 598, the presence of an aortic systolic murmur is no justification for the diagnosis of aortic stenosis, since it may be produced by so many causes. The following cardinal points, when present, render the diagnosis certain: a harsh systolic murmur heard in the aortic area, with a thrill felt at the base of the heart, and a very feeble or absent aortic second sound, a slow small pulse with a heaving ventricle, and increase in vertical direction of the cardiac dulness. The almost constant association of more or less incompetency with the stenosis must never be forgotten, though in the above description the existence of pure obstruction is assumed.

Aneurism is sometimes excluded with difficulty; but the smallness of the pulse, the feebleness of the aortic second sound, the evidence of hypertrophy, and the marked harsh quality of the murmur, are all against aneurism. Moreover, in aneurism there is retardation of the pulse. Babcock attaches importance to the value of the X-rays in the differential diagnosis.

Dilatation of the aorta is rendered probable by the accentuation and purity of the aortic second sound, though Allbutt is doubtful if ever a systolic murmur is present under these circumstances without disease of the orifice.

Prognosis.—Upon the whole the prognosis in aortic stenosis is more favourable than in other valve-diseases; but this is

only true if we include the senile cases, which are often discovered after death from some other affection. In younger subjects the progressive stenosis is apt to terminate life before forty, even when compensation is good. This is in marked contrast to the prognosis of mitral regurgitation. Death may take place suddenly during exertion when the orifice is very narrow, the immediate cause being syncope; sometimes dilatation and pulmonary congestion are the final factors.

AORTIC REGURGITATION

Incompetency, insufficiency, or regurgitation at the aortic orifice is a much more common condition than stenosis, and both conditions are frequently to be met with together.

Etiology and Pathology.—The causes have been duly enumerated in the general description of valvular disease, but it may be here emphasised, that, though rheumatic endocarditis is the most common cause in young subjects, *muscular strain*, *syphilis*, *alcohol*, and the *atheroma* to which they lead, are the more frequently observed factors when the disease appears at a later period of life. In typical cases occurring at middle-life in men (in whom the disease is more common than in women) several or all of the above causes have been obviously operative. Thus, the disease is very frequently associated in a given case with laborious occupations involving continual muscular strain in a syphilitic subject addicted to chronic alcoholism, in which case it may be regarded as a local manifestation of widespread arterio-sclerotic changes.

Independent of age, there is another feature of importance in the rheumatic cases—it is comparatively rare for rheumatic endocarditis to lay the foundation of aortic disease without implicating the mitral orifice at the same time.

Ulcerative endocarditis and rupture of a valve-segment by *sudden* strain are undoubted etiological factors in the production of aortic regurgitation; but the members of this group may, in a sense, be said to be of less clinical importance, since they are liable to rapidly end fatally. The congenital cases are excluded from present consideration.

The *post-mortem* appearances of the valves are such as are to be met with in the lesions affecting the other cardiac orifices; there are thickening, puckering, adhesion of the segments, vegetations along their margins, or fibrotic changes along the line of their attached bases. In the atheromatous cases, as well as in the rheumatic ones, there is often marked calcareous infiltration, which is also often seen in the atheromatous aorta, and which may be the cause of a fatal coronary stenosis. The result of these changes in the valves is that their closure in diastole is necessarily imperfect, and the blood streams backwards into the ventricle till its next systole

occurs ; during systole the deformity often causes more or less obstruction from stenosis.

In some cases the regurgitation occurs with valves whose segments are not deformed, owing to stretching of the fibrous aortic ring, when there is great dilatation of the commencement of the aortic arch, or when an aneurism exists. This is called *relative insufficiency*, and occurs much less frequently at the aortic orifice than at the larger muscular mitral ring when the left ventricle dilates.

Compensation.—The effects which follow aortic regurgitation are already described upon page 590. Enormous dilatation and hypertrophy result, so that the greatest increase in capacity and in weight is reached, the so-called 'ox-heart,' or cor bovinum, which may weigh more than three pounds, being produced. In such an organ all the chambers are dilated and hypertrophied, owing to the effects of the backward pressure already described.

Symptoms.—As in all valvular lesions, when compensation has been silently at work repairing the effects of the valve-injury step by step as the deepening cicatrisation and shortening advances, no symptoms of cardiac embarrassment appear. Even with the enormous enlargement of the heart in aortic regurgitation the patient may be unconscious of ill, the disease being entirely latent.

Usually, however, before the characteristic signs of failing compensation have manifested themselves, nervous symptoms may be observed which are not obviously associated with the cardiac condition. Headache, twitchings, tinnitus, and dizziness, or vertigo, with insomnia, are common, and in a variable percentage of cases mental disturbance, commencing with restlessness and displays of temper, and ending in hallucinations, delusions, and delirium, with suicidal tendencies, not very unlike what is seen in delirium tremens, may be present.

Pain is often felt over the heart, at first commencing in a sense of oppression behind the sternum, usually aggravated by muscular movement, and gradually or suddenly blossoming into true angina pectoris, or anginiform seizures more intense in their severity in aortic insufficiency than in any other valvular disease, the agonising pain radiating down the left arm and towards the neck and back. These attacks may be associated with or alternate with partial syncopal seizures and the belching of flatus.

At a later stage, when compensation begins to fail, the symptoms are more definitely attributable to embarrassment of the circulation. Palpitation, breathlessness, dyspnœa, cough, pulmonary congestion and œdema, hæmoptysis, hepatic enlargement, and dropsy, supervene as the dilatation of the left ventricle increases the pressure in the left auricle by

relative mitral insufficiency, involving the pulmonary veins and finally the right side of the heart (see page 590).

Before this final stage of rupture of the compensatory process, anæmia is often evident, and some degree of wasting, so that the face and lips are usually pallid, contrasting with the dusky lividity of mitral disease. Embolic seizures from the sclerosed valvular foci may terminate life before the final arrest of the wearied-out ventricles ; but sudden death from syncope may occur at any stage of the malady, even before any warning of failure of compensation occurs. Coronary disease, or acute dilatation following exertion, may be the cause of death.

Physical Signs. — During the latent stage the following signs may be noticed :

Inspection.—Bulging of the cardiac area, especially in young subjects, is often very obvious ; it reaches its greatest extent in cases seen in middle-life, where the disease has originated in an attack of rheumatism many years previously. The apex-beat is visible over a large area in the sixth or seventh space, and displaced at the same time outwards, sometimes as far as the middle line of the axilla. When the right ventricle has hypertrophied there will be pulsation in the epigastrium. In great hypertrophy, with much dilatation of the left ventricle, the apex-beat may be replaced by a diffused steady heave visible over a large area of the præcordia.

The great arteries of the neck show markedly exaggerated pulsation, which, however, is also visible in all the superficial arteries of the body. The phenomenon of capillary pulsation may be observed by watching the circulation in the vessels below the finger-nails, or after making an erythematous streak by drawing a line on the forehead with the finger-nail : the red streak intensifies in colour with each pulsation and becomes pallid during diastole. Often a visible pulsation may be observed in the superficial veins, especially in those of the back of the hand when the arm is hanging.

Palpation.—The impulse is felt to be strong, slow, and heaving, even when an apex-beat is not visible. A diastolic thrill is occasionally felt, and not rarely a systolic vibration may be detected.

Percussion.—The area of cardiac dulness is markedly increased downwards and to the left ; and should secondary dilatation have occurred in the right ventricle and auricle, the dulness extends well beyond the right margin of the sternum.

Auscultation.—The most important sign of aortic regurgitation is a long, blowing, diastolic murmur, whose seat of maximum intensity is about mid-sternum, or in the fourth left intercostal space, and whose line of conduction is downwards along the sternum and sometimes towards the apex. As more

or less obstruction often co-exists, a systolic murmur may be heard at the aortic cartilage ; but often this is present where mere roughness of the valve-margins or dilatation of the aortic arch exists independent of narrowing of the aortic orifice. The diastolic murmur is obviously caused by the stream of blood which regurgitates through the imperfect closure of the deformed semilunar valves, and its line of conduction corresponds in the main with the direction of the stream, being more readily conducted to the surface by the osteal tissue than by the ventricular walls, especially as the right ventricle makes up the greater portion of the anterior aspect of the heart.

The aortic second sound may be clear and distinct, but sometimes it is absent. When such is the case, it is supposed to indicate an extensive degree of regurgitation ; the pulmonary second sound is generally normal, and the first sound at the apex may be prolonged and booming or somewhat muffled, and the second sound feeble.

It must always be remembered that, though it may be the easiest ' to time ' when its presence is detected, the diastolic aortic murmur may be missed altogether, owing to its quality of great softness ; and the entire cardiac area should be auscultated if there be reason to suspect aortic insufficiency, the patient being examined in both the erect and horizontal postures. A very soft and almost inaudible murmur may be found, with a high degree of regurgitation. The murmur is usually found immediately to follow the second sound, and it occupies the long pause of the heart ; but sometimes it is not audible for an appreciable interval after the second sound, in which case it is difficult to differentiate it from an auriculo-systolic bruit, save by its quality and by detecting the great hypertrophy of the left ventricle. Occasionally a rumbling presystolic murmur (known as Flint's murmur) is audible at the apex and confined to this region, or a little internal to it ; it is supposed to be produced at the mitral valve by the regurgitant stream striking its anterior flap, and, as a thrill may accompany it, it is often regarded as evidence of mitral stenosis, though *post-mortem* examination usually reveals a normal mitral orifice. During the stage of failing compensation a mitral regurgitant murmur, caused by relative insufficiency, is usually heard, and it may cause the physical signs of the original aortic malady to be overshadowed.

Pulse.—The visible pulsation in the arteries is associated with very marked characteristics when the finger-tips are applied ; these constitute the so-called ' water-hammer ' or Corrigan's pulse. The vessel during diastole feels to the finger as if empty, but each ventricular contraction gives rise to the sensation as if a little ball of blood was suddenly shot along the vessel ; the arterial coats collapse as suddenly as

they had previously been distended. The effect is heightened if the patient's arms are elevated above his head whilst the finger is applied to the radial artery, the thud being then very perceptible, even when the arterial coats are considerably thickened. As a rule, unless in failing compensation, the pulse is regular, and, according to Broadbent, slightly retarded; irregularity of the pulse is always a serious symptom in aortic disease.

By pressing moderately with the edge of the chest-piece of the stethoscope upon the femoral artery, a double murmur is produced (Duroziez's sign). The radial pulse-tracing will show a high vertical up-stroke caused by the exaggerated percussion-wave, with a sharp apex and short tidal wave, and a very small dicrotic or recoil wave. More or less arterial sclerosis will be found in all long-standing cases, even of rheumatic origin, owing to the repeated hammering of the enormously hypertrophied ventricle on the vascular walls.

Diagnosis. — The recession of the tissues between the apex and the sternum, caused by atmospheric pressure when the dilated heart diminishes its bulk in systole, may simulate *adherent pericardium*, but the presence of the characteristic pulse, diastolic murmur, and the downward displacement of the movable apex-beat show that the case is one of aortic disease.

Dilatation of the aorta, if not also associated with disease of the valves, may be distinguished by the ringing metallic character of the second sound in the aortic area.

Allbutt lays stress upon the *quality* of the murmur in those cases where *mitral regurgitation* is simulated; the chances of a distinctly musical bruit being aortic he calculates as four to one, and if the murmur is heard at a distance from the chest-wall it is almost certain (twenty to one) to be aortic. The characters of the pulse in such cases are of great value.

Mitral stenosis closely simulates those cases of aortic regurgitation where the murmur is only audible towards the end of the long pause, but in aortic disease the site, the soft and blowing quality, and the direction in which the adventitious sound is communicated, together with the pulse-characters, settle the diagnosis. The presence of Flint's murmur, which occurs in nearly half the cases of aortic regurgitation, is no evidence of mitral stenosis. Its rumbling quality, taken together with its erratic disappearances and returns, the water-hammer pulse, hypertrophy of the left ventricle, and the typical diastolic aortic murmur, also always present, demonstrate the true nature of the case. Moreover, the snap of the first sound, which invariably terminates the murmur of mitral stenosis, is absent with Flint's murmur.

At a late stage of failing compensation, the presence of relative mitral insufficiency changes the entire clinical picture

by the advent of dyspnœa, venous engorgement, dropsy of the legs, hepatic enlargement, &c. ; but this change causes little difficulty when read in the light of the previous history.

Prognosis.— This is always serious, aortic regurgitation being the most formidable and grave of all the valvular affections. Age modifies the outlook ; in young subjects compensation reaches its highest degree, and the disease may remain latent for many years unless other pathological processes set in, as during protracted fevers, influenza, diphtheria, &c.

Rheumatic cases have a much longer duration than sclerotic ones, and Osler rightly gives the first place in prognosis to the condition of the coronary arteries. These may escape for many years when the origin is rheumatic, but they are involved early where the valvular disease is but a part of a general arterio-sclerosis. The advent of anginal symptoms is therefore one of the gravest omen. The writer finds in his experience that the prognosis deepens with the number of contributory agents at work in a given case ; thus, in an intemperate patient, whose occupation involves severe muscular labour, and who has suffered from syphilis, the duration of life may certainly be estimated as short—shorter still if arterio-sclerosis be associated with renal changes.

Allbutt's experience is perhaps more gloomy than that of most observers : he states that he cannot remember the survival of any patient with aortic regurgitation for a period of fifteen years. Doubtless, however, many of his cases had been of considerable standing before coming under observation. Babcock relates one case where the disease existed in a severe form for over twenty-eight years.

Cases of rupture of a cusp from sudden violent strain are always most grave ; and if there be evidence of previous degenerate change, a rapidly fatal event may be expected.

The probability of sudden death must be always remembered. This may be brought about in any of the ways already mentioned, and it may occur during the strictly latent period before any symptom has been observed, or it may occur during the stage when pain and precordial oppression are giving their warning that failure of compensation is about to follow ; death is more common in this latter stage of ruptured compensation.

The treatment will be discussed later on.

MITRAL REGURGITATION

Etiology and Pathology.—Insufficiency or incompetency at the mitral orifice is the result of the etiological factors already detailed in the general description of valvular disease. By far the most common cause is the endocarditis accompanying acute rheumatism and chorea, the cicatrisation of

the inflammatory exudation producing such deformities in the mitral curtains as prevent their perfect closure during ventricular systole, so that a quantity of blood is driven backwards into the left auricle.

The chordæ tendineæ and papillary muscles participate in the general gluing, thickening, and shortening, which is further exaggerated by the growth of vegetations on the valve-margins, and the deposit of lime-salts at their bases, so that very often the result is a certain degree of obstruction as well as of incompetency.

Other causes, as rupture of a valve-segment, ulceration of the endocardium, fibroid degeneration of the myocardium, even when not associated with dilatation, are of less importance.

Relative insufficiency is a common cause of mitral regurgitation, as mentioned under 'Aortic Incompetency' and 'Degenerations of the Myocardium.' The condition in these cases is the result of a degree of ventricular dilatation which stretches the mitral ring so that the segments of the otherwise healthy valve are unable to reach across the chasm and close it. This state is also met with in the failing compensation supervening in chronic Bright's disease which has produced hypertrophy of the left ventricle; it is occasionally also seen in the weakening of the cardiac muscle following protracted fevers, in anæmic states, alcoholism, and in the tachycardia of Graves' disease.

Compensation.—The immediate effect of the lesion is to produce dilatation of the left auricle, since this chamber, in addition to the charge of blood which it receives from the pulmonary veins, must also contain the regurgitated quantum forced into it by the ventricle; hypertrophy follows. The left ventricle receiving this increased amount of blood from the hypertrophied and dilated auricle also dilates and hypertrophies. The increased pressure in the enlarged auricle leads to obstruction in the return of the blood from the pulmonary veins, to overcome which the right ventricle, and finally the right auricle, dilate and become the seat of hypertrophy.

Symptoms.—The natural compensatory changes just mentioned proceed at the same time that the cicatricial deformity is gradually interfering with the competency of the valve, so that the patient is unconscious of any embarrassment. Should, however, rupture of a valve-segment occur, grave disturbance rapidly supervenes, which may prove fatal before the natural process has time to adjust the balance, and this is not a rare phenomenon in ulcerative endocarditis.

After compensation has been firmly established, the valvular mischief, if the patient leads a uniformly quiet life, may remain latent for many years, during which no symptom may awaken the patient's suspicions, unless he should chance

unfortunately, perhaps, to come under the observation of a physician who is likely to prescribe digitalis. Owing to the diminution of the cardiac reserve power, as already described, any sudden severe demand upon the heart may elicit evidence of a damaged organ by the production of cardiac pain, dyspnœa, or palpitation, which, however, usually rapidly pass off.

A stage is generally ultimately reached in which, owing to some severe intercurrent illness, impaired nutrition, cardiac over-strain, or the advance of age, the muscular power of the left ventricle begins to fail, and the chamber slowly dilates. Then the symptoms already described upon page 593 gradually develop—*dyspnœa* upon exertion, with more or less *palpitation*, *cough*, and evidence of *venous stagnation*, as seen in lividity of the lips, ear-tips, and cheeks, and clubbing of the finger-ends.

Bronchial catarrh and, at a later stage, hæmoptysis, œdema of the lung, and hydrothorax supervene.

The dyspnœa, which was only noticeable on exertion at first, becomes, especially at night, paroxysmal or continuous, and, as the venous stagnation increases, cyanosis appears, the liver and spleen swell, slight jaundice and ascites occur, and dropsy, beginning in the legs, soon invades the trunk. Albuminous, and often bloody, urine results from the renal congestion, or desquamative nephritis sets in.

In long-standing cases, where the venous stasis in the internal organs has been marked, the *post-mortem* results correspond with the clinical features of the case during the later end of life; thus brown induration of the lungs and hepatic, renal, and splenic engorgement, or even secondary atrophic changes, may be observed.

Under rest and appropriate treatment the balance may again be adjusted, and by a quiet life and cardiac tonics the dilatation may give way to hypertrophy.

Physical Signs.—During the period of compensation the following signs are present on examination; at a later stage they are also to be made out, but more or less masked by the pulmonary and other engorgement:

Inspection.—The patient's appearance is normal, but there may on close inspection be observed some heightened colour, due to slight capillary congestion, which later on deepens into lividity or cyanosis. In young subjects there is usually some bulging or prominence of the lower precordial region. The apex-beat is displaced downwards and to the left, and may be seen as far outwards as the anterior line of the axilla; it may be visible over a considerable area. At a later stage, when compensation is failing, it may be wavy and diffuse, and associated with jugular and hepatic pulsation. There is usually epigastric pulsation, which is indicative of hypertrophy of the right ventricle.

Palpation.—With perfect compensation the impulse will be strong and heaving, and by the touch some idea of the amount of hypertrophy of the left and right ventricles may be obtained. A systolic thrill at the apex is rarely felt, but a presystolic thrill, indicative of associated stenosis, is often present. Epigastric pulsation below the ensiform may be both visible and palpable—the result of the heaving produced by the hypertrophied right heart.

Percussion.—The area of dulness is much increased; it extends outwards as far as, or slightly beyond, the apex, and even when no impulse is visible or palpable it may extend to the axillary line. It must be remembered that the increased dulness is chiefly caused by enlargement of the right ventricle, only the outward margin representing the left ventricle, The upper limit of cardiac dulness may reach the level of the second rib, owing to the hypertrophy of the left auricle, and the right margin will usually be found to extend an inch and a half or more beyond the right of the sternum. Percussion will also reveal increase in the vertical dulness over the liver, whose lower margin, in failing compensation, will be well below the costal arch, where it can be palpated.

Auscultation.—A soft blowing murmur is heard over the apex of the enlarged heart; it is best described by Sansom, who likens it to a whispered 'who.' It is usually said to often possess musical qualities, but a *distinctly musical* murmur is uncommon in mitral regurgitation. Even when it is but a short 'whiff,' it never ends abruptly, but fades off into silence. It may entirely replace the first sound, but more frequently it accompanies it, and may be audible all through the short pause. Its site of maximum intensity is just over the apex, though it may be audible all over the præcordium; its line of conduction is outwards towards the left axilla, and it is often distinctly heard close to the spine at the level of the scapular angle. The writer has heard it several times in this latter situation when it was inaudible over the heart.

Occasionally it is only heard along the left edge of the sternum from above the ensiform to the level of the second cartilage. The first sound, as stated, may be absent, its place being taken by the murmur; but usually as much of its valvular character may be discerned as will assist in the diagnosis of the time or rhythm of the bruit, which is always systolic, occurring at the moment when the apex strikes the chest-wall. The second sound at the pulmonary area is clear and accentuated; it may occasionally be absent at the apex, nothing being audible but the succession of whiffs. The disappearance of the accentuation of the pulmonary second sound at the base is an indication that the muscular power of the right ventricle is failing.

The murmur of mitral regurgitation is occasionally influenced by changing the patient's position: it sometimes disappears on assuming the erect position, though Potain maintains that when this occurs it may be taken as evidence that the murmur is cardio-pulmonary—*i.e.* one produced by the ventricular contraction drawing the air into the alveoli in the adjacent portions of the lung, which had been compressed during diastole. Sansom points out, however, that Potain's murmur is not heard over the exact apex, but close to the right or left of it, and that it never replaces, but follows the first sound.

When mitral stenosis is a marked feature a presystolic bruit will also be present at the apex, and sometimes a tricuspid systolic one at the ensiform when regurgitation occurs as well at this orifice.

The pulse is regular, small, and weak; the tension being low; it is easily obliterated by moderate pressure when the degree of incompetency is extensive, but in fully compensated cases of moderate extent the pulse is practically normal to the touch. The advent of failing compensation is marked by irregularity or arrhythmia, which may be the only marked feature in the sphygmogram.

Diagnosis.—The chief points have been already dwelt upon, the most important being the presence of a systolic apical murmur conducted towards the spine and associated with a loud accentuated second sound heard over the pulmonary area in a heart whose ventricles show a considerable amount of hypertrophy with dilatation.

Aortic regurgitation, when seen in its late stage, in which the enormously hypertrophied chamber has become dilated owing to the failure of its muscular power, may be mistaken for a case of primary mitral regurgitation. The dilatation has caused relative insufficiency; a murmur at the apex with all the characteristics of the mitral bruit is present, and the adventitious sound indicative of aortic insufficiency may be masked by it. In this case, though mitral regurgitation is present, the mitral curtains are normal. The history of the illness is sufficient to clear up all difficulty.

Bright's disease with arterio-sclerosis, *fatty and fibroid degeneration of the myocardium*, *alcoholic heart*, and the dilatation occurring in *anæmia*, all bring about the condition of relative insufficiency in which the mitral segments are intact. The diagnosis of this relative insufficiency from that produced by the puckering of old valvular disease is much simplified by recognising that in its essential features it is nothing more or less than a manifestation of loss of muscular power of the left ventricle, as pointed out by Steell, who maintains that the incompetency is identical with that occurring at the tricuspid under normal conditions. It should be remembered that

the relative insufficiency of anæmia entirely disappears under treatment directed to the blood-condition.

The diagnosis of mitral regurgitation caused by valvular disease or relative insufficiency from those cases in which Potain's cardio-pulmonary murmurs are heard near the apex has been already dealt with in the paragraph under 'Auscultation' (page 610); moreover such murmurs are not associated with hypertrophic changes or with accentuation of the second sound in the pulmonary area.

The presence of a presystolic murmur, it is needless to remark, greatly strengthens the diagnosis of mitral regurgitation caused by valvular disease; and some authorities maintain that a degree of obstruction must exist in every case.

Prognosis.—The outlook is better than in any other valvular disease. When compensation has occurred in youth, the balance may, under favourable conditions, be maintained for a long lifetime; hence the physical signs without any symptoms are often met with in advanced age. In these cases some degree of narrowing is always present.

Even when symptoms of failing compensation set in, by rest and appropriate treatment the balance may be again restored and the patient's life prolonged under conditions often of comparative comfort. The prognosis, however, is always grave when evidences of sclerotic change supervene in the arteries, and especially in the coronary vessels, as age advances, or when syphilis is acquired. In a small percentage of cases in children and young adults the physical signs may entirely disappear after having persisted for several years. The writer has been fortunate in witnessing this result in at least three typical cases.

MITRAL OBSTRUCTION

Etiology and Pathology.—Stenosis or obstruction at the mitral orifice, exclusive of the rare cases of congenital origin, is nearly always the result of an endocarditis following acute rheumatism or chorea produced by the rheumatic coccus. In many cases, however, the history of rheumatism is absent, probably because its manifestations have been overlooked in infancy or childhood. In a very small percentage of cases the disease originates in adults in a general arterio-sclerosis, the result of syphilis, or it may be caused by an endocarditis occurring in association with the contracted kidney of Bright's disease. It is a disease of early life, contrasting in this respect markedly with aortic affections; and girls and women are much more frequently affected than males, the proportion being more than two to one.

The pathological process is essentially the same as that which is operative in other valvular lesions, the inflammatory

exudation into the substance of the valve becoming the seat of fibroid proliferation. The tendency of the newly formed fibrous tissue to contract after cicatrisation is complete leads to valvular deformity. The ventricular ring, musculi papillares, and chordæ tendineæ, share in the same shortening, thickening, and calcification which affect the valve-curtains; in young subjects vegetations are often present.

Two well-marked types of mitral stenosis are recognised—*i.e.* (1) the *funnel* form; (2) the *button-hole*. The former is much more common in childhood, whilst in the adult the button-hole variety is nearly always found. When the margins of the two mitral segments become fused together by the shortening of the new fibrous tissue, the result is a smooth funnel-shaped membrane, the wide mouth of which is situated at the ring, and the small end containing the narrow aperture, owing to the shortening of the chordæ tendineæ, is drawn down towards the apex of the ventricle. Upon opening the left auricle and viewing the deformity from above, the interior of the hollow cone is visible, with its smooth polished wall ending in a rounded opening which may be too small to admit the tip of the little finger.

The button-hole form is produced likewise by the fusion of the margins of the curtains, which are greatly thickened and often calcified, forming a narrow slit-like aperture leading from the auricle into the ventricle. Often both types exist together, the funnel-shaped valve having a narrow slit at its lower extremity instead of a rounded opening.

Other cases are met with where the outline of the narrowed opening is rendered irregular by the projection inwards of calcareous plaques from the ring, the valve-margins being unaffected. Campbell maintains that the narrow slit permits of a much larger current of blood passing through than does an equal area of rounded opening.

An examination of a large number of specimens produces the conviction that regurgitation must almost invariably have also occurred through the stenosed opening. This consideration, however, need not create any difficulty in accepting the fact that the stenosis is the main factor in producing the clinical picture, since the smallness of the regurgitant stream must play a very minor part in the production of signs and symptoms during life, and since also there are grounds for believing that regurgitation under certain conditions is prevented or lessened by the auricular hypertrophy.

The left auricle is dilated and hypertrophied generally to a very obvious degree. White thrombi are often found in its greatly hypertrophied appendix; and sometimes a free ball-thrombus occupies a considerable portion of the general auricular cavity. The endocardial lining of the auricle is

thickened, and at places warty or polypoid vegetations may be found adhering to it, especially in the vicinity of atheromatous patches; hence the frequency of embolism. In some cases the auricle fails to respond to the increased demands made upon its scanty muscular tissue, and no hypertrophy follows.

The pulmonary veins are usually much dilated owing to the increased blood-pressure in the auricle, and from the same cause the right ventricle dilates and hypertrophies. Finally the right auricle does likewise. The tricuspid ring is not uncommonly found widened, and the venæ cavæ dilated. The left ventricle is little affected; often it is smaller than normal, and may be sometimes, as stated by Babcock, almost rudimentary in appearance. Those cases where it is found dilated and hypertrophied, Sansom explains, are the result of pericardial adhesions or arterio-sclerosis; but the explanation is perhaps much simpler—the dilatation and hypertrophy are probably the direct result of a larger degree of regurgitation than usual. The extent of the hypertrophic change in the left ventricle may be accepted as a measure of the extent of the regurgitation which is always present, though Samways maintains that, as just mentioned, the great hypertrophy of the auricle prevents regurgitation, its systole continuing till the opening of the aortic valves. Sansom accepts this as the explanation of the absence of hypertrophy of the left ventricle in the great majority of cases, and as the explanation of the fact that in the last stages of stenosis a regurgitant murmur may appear from failure of the auricle.

Pulmonary infarction and brown degeneration of the lungs are often observable, and corresponding changes are found in the liver, spleen, stomach, intestines, and kidney.

Embolism of the various organs is much more common in mitral stenosis than in other valvular lesions.

In a considerable percentage of cases pulmonary tuberculosis supervenes, probably from the general failure in nutrition caused by a diminished blood-supply to the different organs, or from a diminution or interference with the local blood-supply to the lungs. In children a considerable degree of stunting and wasting is often observed, and clubbing of the finger-tips is nearly always present from enlargement of the terminal phalanges.

Compensation.—As interpreted by the *post-mortem* changes, the stenosis causes hypertrophy and dilatation of the chamber behind it, so that the enlarged auricle in very mild degrees of stenosis may possibly for a long period be able to overcome the slight obstruction without implicating any other part of the heart. Ultimately in the great majority of cases the right ventricle is called upon to exert greater force than the normal in order to overcome the increased pressure in the

pulmonary veins; dilatation with hypertrophy follows, and thus the balance is kept adjusted.

Compensation fails when the dilatation of the right ventricle reaches a stage which produces relative incompetency of the tricuspid valve, the blood during its systole being regurgitated into the right auricle (which may have already become hypertrophied). Venous engorgement follows, with enlargement and pulsation in the liver, and some degree of ascites. Sometimes compensation fails owing to the incomplete hypertrophy of the auricle, as the auricular cavities containing less muscle relatively than the ventricles are more prone to dilatation.

Symptoms.—As in other valve-lesions, when compensation has been established mitral stenosis may remain latent for long periods, but usually the latent stage is much shorter than in mitral regurgitation. In children, owing to the diminished supply of blood sent out from the left ventricle at each stroke, there may be general stunting of the body or wasting, which not rarely ends in phthisis; anæmia is common, especially in girls, who are prone to suffer from severe chlorosis, and often the mental development is retarded.

During the latent period various symptoms may suddenly exhibit themselves in cases where no heart-lesion was previously suspected. These are the results of embolism, which may produce hemiplegia, not rarely associated with aphasia; pulmonary infarction from blocking of a pulmonary vessel may produce sudden dyspnœa, cough, consolidation of the lung, and hæmoptysis.

Chorea is a common event, and establishes the rheumatic origin of the original stenosis. In such cases there is probably fresh inflammatory endocardial vegetations—a result very common in mitral stenosis, which leads to further crippling of the deformed valve.

These recent vegetations may be followed by the arterial pluggings characteristic of ulcerative endocarditis, which are clinically different from the aseptic blockings caused by the detachment of fibrinous plugs formed in the dilated chambers of the heart which commonly cause infarction and hemiplegia.

As in other valve-lesions, during the latent period severe muscular exercise, as hill-climbing or running, may bring on dyspnœa, palpitation, and precordial distress, which, however, pass away after rest; but even in compensated cases breathlessness is often produced by trivial causes. Attacks of epistaxis, more common at a later stage, may occasionally be noted at this period. Subjects with mitral stenosis are more prone to bronchial attacks on slight exposure to cold and damp or to vitiated atmospheres.

Failure of compensation finally makes its appearance at a much earlier age generally than in mitral regurgitation, and with symptoms of similar type, though some differences

are noticeable. Dyspnœa, cough, pulmonary engorgement, lividity, hepatic enlargement, and marked irregularity in the heart's action (which is always greatly accelerated) are commonly observed. But general dropsy is much less frequently present than in mitral insufficiency, probably because many patients die from failing power of the left auricle, which cuts short the train of symptoms depending upon tricuspid insufficiency ; some degree of ascites is, however, often evident.

During the stage of failing compensation any of the accidental phenomena noticed sometimes in the latent period, as hemiplegia, aphasia, and infarctions, may suddenly appear and terminate life with tragic suddenness—an event comparatively rare in mitral insufficiency.

In the small group of cases originating in general arterial sclerosis and fibroid kidney the symptoms peculiar to the original disease are blended with those of failing compensation ; thus an extensive apoplexy caused by cerebral hæmorrhage, or uræmic convulsions may abruptly terminate life or call attention for the first time to the mitral lesion.

Many authorities describe two stages in mitral stenosis. In the first stage the pulse is regular, and in the second there is marked irregularity. Whilst this latter sign is commonly regarded as evidence of failure of compensation, Hay maintains that the irregularity may be due to other causes, and is not to be accepted as a guide to the degree of cardiac failure or stenosis present. He points out that patients may die from progressive cardiac failure with œdema, orthopnœa, &c., though the pulse may remain quite regular, and also that there may be marked irregularity with fair compensation. Mackenzie has shown that the difference between the two conditions depends upon an alteration in the manner in which the stimuli for contraction are conducted. In the normal sequence of events the auricles contract first just before the ventricles, but in the so-called second stage the stimulus starts in the nodal thickening of the bundle of His, and radiates simultaneously both upwards to the auricles and downwards to the ventricles, causing what he designates as the nodal or disorderly rhythm. Hay has shown that this nodal rhythm or arrhythmia is readily slowed by digitalis, which possesses very little power in reducing the frequency of the quick, regularly beating heart, in the first stage of the stenosis.

Mackenzie emphasises the fact, frequently overlooked by clinicians, that the crescendo murmur of mitral stenosis is never heard after the heart has taken on the nodal or disorderly rhythm.

Physical Signs.—During the period of compensation the following signs are present on examination :

Inspection.—Sometimes, especially in children, there is prominence or bulging over the enlarged right ventricle about

the ensiform region. The apex-beat may be normal, absent, or diffused ; rarely is it much displaced beyond the nipple, since enlargement of the left ventricle only occurs in a small percentage of cases, and the part of the heart in contact with the chest-wall is right ventricle. When this is markedly hypertrophied an obvious pulsation or heave is visible below and behind the ensiform ; and if with this there be no evidence of an impulse beyond the normal apex-region the sign is of great diagnostic value in the case of children with thin chest-wall.

Often a pulsation is visible in young subjects at the base of the heart in the third left interspace between the costal cartilages. This impulse, when contrasted with that of the apex, is found to precede it, being synchronous with the contraction of the auricle. It may be associated with an auriculo-systolic pulsation in the jugular vein immediately above the right clavicle.

An examination made in the stage of failing compensation may reveal visible pulsation caused by tricuspid regurgitation in the neck-veins during ventricular systole.

Palpation.—By placing the hand over the precordial region, (*a*) the heaving impulse of the hypertrophied right ventricle is felt to the left of the ensiform cartilage ; whilst (*b*) the absence of pulsation to the left of the normal apex-spot is confirmed. (*c*) A well-marked thrill is felt just over the apex or slightly to its right ; this conveys to the hand a sensation of ' purring,' which may be soft or harsh, and may be long or short, sometimes commencing early in diastole, at other times just before systole, but in all cases it ends abruptly with the impulse-beat. In the absence of aortic disease this presystolic thrill is pathognomonic of mitral stenosis ; it corresponds to the characteristic presystolic murmur in the varieties of its rhythm and in its abrupt termination. (*d*) The pulsation in the third left interspace, already observed, on inspection may be timed by comparison with the thrill ; it is synchronous with this and found to precede the apex-beat. (*e*) By placing the hand over the precordial region so that the finger-tips fall below the right costal arch pulsation of the liver synchronous with the apex-beat may be felt in the stage of failing compensation in some cases. In the search for the characteristic thrill the patient should be examined in the lying-down and sitting-forward positions, and before and after gentle exercise.

Percussion.—This serves to confirm the limits of enlargement already tested by sight and touch. The transverse area of cardiac dulness may extend more than one inch beyond the right margin of the sternum to the line of the left nipple, but when regurgitation is marked the left margin of dulness may extend to the anterior line of the axilla.

It will be observed that the extension of the cardiac dulness to the right and downwards is often greater than in regurgitation.

Auscultation.—The chief sign of mitral stenosis is the presence of the so-called 'presystolic' murmur, whose features in all cases require systematic investigation. As regards its *quality*, which is a very important character, it is usually rough or harsh, though often described as rumbling, purring, snoring, vibratory, &c. These features are, however, of little importance when compared with its crescendo character. No matter what its other qualities may be, or how long or short may be its duration, it usually increases in intensity, but always stops abruptly with a snap. This snap is supposed to be the short accentuated first sound of the heart in which the murmur always ends.

Its point of maximum intensity is at or immediately internal to the apex, and it is often strictly localised over a small area in this region. Though very occasionally heard over the entire cardiac region, it cannot be said to possess any line of conduction, and it is very rarely heard at the back.

Its time or rhythm is before the first sound; hence its usual name—presystolic—though presently the correctness of this title will be discussed. Its rhythm is easily determined by its relation to the altered first sound, with which it blends. It may also be demonstrated to be synchronous with the apical thrill, and not with the apex-beat. These facts, first pointed out by Sir W. Gairdner, whose observations had all the importance of a discovery in the region of physical diagnosis, suggested to him the name of *auriculo-systolic* murmur.

The factors in the production of this murmur have been always the subject of keen debate. The usually accepted view is that the adventitious sound is produced by the current of blood passing over the margins of the constricted orifice, and that its crescendo character and abrupt termination is caused by the sudden systole of the auricle which immediately precedes the first sound. Sansom states in his exhaustive article in Allbutt's 'System' that 'the evidence leaves no room for doubt that the reinforcement towards the close is coincident with and due to the muscular contraction of the auricle.'

All authorities point out that the murmur is rarely, if ever, of a blowing character. That its essential quality consists in its being a modified blowing sound is capable of easy demonstration by the following simple experiment. When a current of air is being blown through the partially closed lips, if the palm of the hand be made to strike the mouth *very gently* for a number of times, a series of exact reproductions of the presystolic murmur is obtained. The crescendo quality and sudden termination are produced by the meeting of the two

currents of air just before the oral aperture is closed by the palm. On increasing the force of the current produced by the movement of the hand, and on blowing more strongly, the effect is intensified. If we contrast this simple procedure with that which is taking place in the heart of mitral stenosis a feasible explanation of the production of the crescendo character of the presystolic murmur may probably be forthcoming. The fluid vein caused by the column or jet of blood passing over the stenosed aperture meets the volume of blood which is forced upwards against the lower surface of the funnel or button-hole by the systole of the ventricle. This simple explanation of the crescendo character of the presystolic murmur receives support from the recent researches of Mackenzie, which show that the crescendo feature disappears when the disorderly rhythm sets in, owing to simultaneous contraction of the auricle and ventricle.

Four varieties of the murmur indicative of mitral stenosis are met with. If, instead of calling the murmur presystolic, the term *diastolic* be substituted, the nomenclature is simplified. The bruit just described is only one, but the most important one, of these : since it precedes the first sound and ventricular systole it is strictly *diastolic—i.e.* it is produced during the period of ventricular diastole.

When the murmur immediately precedes the first sound it is said to be *late diastolic*. This is the short presystolic or auriculo-systolic bruit just discussed. When it begins immediately after the second sound and runs all through diastole and auricular systole, ending with a snap in the first sound, it is of long duration, and is called an *entire diastolic* murmur.

When the murmur is short, and immediately follows the second sound, it is said to be *early diastolic*. When occurring as an isolated bruit between the second and first sounds it is called *middle diastolic*. The production of these latter murmurs, which are blowing in character, is caused by the flow of blood entering from the pulmonary veins over the margins of the stenosed aperture when the auricle is passive or in diastole. The flow is aided by the suction-power of the left ventricle during its dilatation in diastole. As soon as the auricular systole occurs, the crescendo quality is superadded. When the murmurs tail off, as the early and middle diastolics do, without abrupt termination, the muscular power of the auricle is probably failing. Such murmurs may be converted into the long or entire diastolic variety by brisk exercise.

The above nomenclature is too refined ; and for all practical purposes these murmurs may be regarded as mere modifications of the typical auriculo-systolic, and should be grouped as the presystolic murmur.

Accentuation of the second sound in the pulmonary area is of great importance. Though generally not so distinctly marked as in mitral regurgitation, it should be carefully searched for, as it is indicative of a high degree of pressure in the pulmonary circuit.

Reduplication of the second sound, so-called, is the sign sometimes styled the 'postman's knock,' which may be heard at the apex or base. Sansom maintains that the 'rat-tat' is not a true doubling of the second sound, whether heard at base or apex, but that the first element of the double sound is the normal second sound, chiefly pulmonic, and the following knock is a tension sound, and is produced at the mitral valve by the inrush of blood through the narrowed orifice into the ventricle.

Whilst this explanation may hold for the so-called reduplication of the second sound when heard at the *apex*, it is still maintained by most authorities that the doubling of the second sound in mitral stenosis, which is much more frequently heard at the *base*, is a *true* reduplication. The most generally accepted theory for its explanation is that it is due to asynchronism in the closure of the aortic and pulmonic valves. This failure in synchronism is believed by Barr, Broadbent, Babcock, and others to be due to the difference in pressure of the blood in the aorta and pulmonary artery, which causes the respective valves to close earlier or later. Barr believes, contrary to other authorities, that the increased pressure causes delay in closure of the pulmonary valves, as the ventricle contracts more slowly to overcome the resistance. Sansom argues that, whether heard at apex or base, the doubling is only apparent, and that his explanation just mentioned holds for the double sounds heard at both apex and base.

Reduplication of the first sound is very occasionally heard. It has been variously attributed to asynchronism of the ventricles, irregularity of the action of the musculi papillares, and the presence of a hemisystole.

The sounds differ from the triple rhythm of the galloping bruit caused by muscular failure.

A *mitral regurgitant murmur* is audible at the apex in a considerable number of cases where the presystolic is also present. It is caused by the patency of the narrow opening, which sometimes admits of enough regurgitation to produce a bruit during ventricular systole.

Absence of the second sound of the heart at the apex. This is a sign of advanced disease, and is usually associated with marked shortening and intensification of the first sound, which, with its presystolic murmur, is alone audible. The cause of the absence of the second sound at the apex is held by Broadbent to be owing to the hypertrophy of the right

ventricle, which occupies the anterior portion of the heart and prevents the aortic element being conducted to the surface. It is this aortic element of the second sound which is heard in the normal condition at the apex. Hence Sansom is probably correct in maintaining that when there is a second sound heard at the apex it is the pulmonic second.

Disappearance of the presystolic murmur, due to failure of auricular power in the late stages of failing compensation, is a common event. When this happens, nothing is heard at the apex but the short, sharp, first sound of the heart.

The sharp snapping character of the first sound has been variously explained, some regarding it as due to abbreviated systole of the left ventricle, whilst others consider that it is produced at the tricuspid. It is just possible that it is an adventitious sound produced by the contact of the two columns of blood, as mentioned in the writer's explanation of the abrupt termination of the presystolic murmur on a previous page.

Broadbent, by means of the two last-mentioned physical signs, divides the progress of mitral stenosis into three stages: (1) That in which at the apex there is a presystolic murmur, a first and a second sound; (2) when there is a first sound and presystolic murmur; and (3) in which only the short, sharp sound is audible. This clinical division is, however, often useless, since one of the chief features in the disease is the changeability of the presystolic murmur, which is apt to disappear and return in a capricious manner.

In this third and last stage, however, it is not rare for a systolic apical murmur to show itself for the first time, which is due either to mitral or tricuspid regurgitation caused by relative insufficiency.

Irregularity in the rhythm and force of the heart is a more common sign in mitral stenosis than in most other heart-affections of structural origin; it does not necessarily belong to the stage when compensation is failing. (See page 615.)

The *pulse* in the earlier stages is regular and small and of fair tension, though irregularity in force is not rare even in the stage of complete compensation. The pulse may be markedly irregular, whilst auscultation reveals a regularly acting heart, some of whose beats fail to influence the radial artery.

The sphygmogram may demonstrate an irregularity not perceptible to touch or hearing. This is seen in a number of small elevations in the long sloping down-stroke. When the tension is unusually high from peripheral resistance the up-stroke is short and the pulse may be anacrotic, but often the up-stroke is longer, indicating an imperfectly filled condition of the artery.

Diagnosis.—The typical presystolic murmur ending in the sharp snap of the first sound, with the presence of a pre-

systolic thrill, evidence of hypertrophy of the right ventricle, and accentuation of the pulmonary second sound, render the diagnosis during the compensation stage a matter of little difficulty.

Aortic regurgitation, however, is sometimes mistaken for mitral stenosis, owing to the presence of Flint's presystolic murmur; the differential diagnosis is discussed upon page 605. In a very considerable number of cases, however, both aortic insufficiency and mitral stenosis occur together, in which condition the difficulty in diagnosis is very great and may amount to an impossibility when the case comes for the first time under observation after compensation has broken down. A careful search for the physical signs produced by each lesion in the heart and blood-vessels will usually clear up the diagnosis.

Tricuspid regurgitation, from primary valvular disease, may be eliminated in most cases by its extreme rarity; but its existence may be suspected when the site of the murmur is at the fourth left intercostal space, and when, in the late stage, there is great cyanosis, general anasarca, and jugular pulsation. As already explained, it is a common sequel to dilatation, in which case it is produced by relative insufficiency.

Adherent pericardium is sometimes associated with a murmur resembling that of mitral stenosis; the presence of such signs as systolic retraction of the tissues at the apex and of the lower end of the sternum and adjacent ribs on the left side, and of diastolic rebound and collapse of the neck-veins, will serve to prove the nature of the case in most instances.

Mitral regurgitation may be safely diagnosed as associated with the stenosis when a systolic murmur accompanies the first sound in the compensation-stage, the double murmur being characteristic, with the short snap between. When, however, the second sound is absent at the apex in pure stenosis, and only a long presystolic murmur and short first sound are audible, the latter may be mistaken for a second sound preceded by a systolic murmur; but the *quality* of the presystolic murmur—its crescendo character—should always be sufficient to distinguish it.

In the stage of advanced failure of compensation, when the auricle fails to prevent regurgitation by its loss of muscle-power (which enables it to close the opening in the earlier stages of the stenosis), then mitral regurgitation occurs, and, the presystolic murmur being abolished, a false view of the primary lesion is inevitable, unless the natural sequence of events be kept in mind to explain the late first appearance of the systolic murmur.

Prognosis.—This, though not so grave as in aortic regurgitation, is very much more serious than in the remaining valvular lesions, probably because the nature of the stenosis, which

submits the margins of the valve to continual friction and strain, is more likely to be progressive, as suggested by Broadbent, on account of the resulting chronic inflammation.

This also accounts for the fact that the prognosis is worse where the disease becomes established in very early life, in which case the narrow orifice remains stationary or gets smaller, while the heart increases in size to meet the increased demands of the body-development.

The duration of life is greatly influenced by fresh attacks of endocarditis, which are very prone to occur in subsequent rheumatic seizures. Under 'Endocarditis' is recorded a case where ulcerative endocarditis supervened under the writer's observation, the narrow slit having ulcerated at one of its extremities, so that an extensive regurgitation suddenly took the place of an aggravated stenosis, as proved by *post-mortem* examination.

The absence of the second sound at the apex may be usually accepted as a warning that failure of compensation is likely to occur at any time, though a patient may live for a considerable period without much embarrassment when this condition exists. Marked irregularity of the pulse is always a serious sign.

The disappearance of the presystolic murmur is usually the herald of a final breakdown; but when it follows upon the appearance of some passing strain or transient illness, by rest and other measures the murmur may reappear, and compensation be again restored for a further period.

The presence of embolism and of infarction obviously increases the danger to life, and this is more liable to occur in pregnancy, so that when this complicates the case the prognosis is serious or grave. Marriage should therefore be discouraged or forbidden in mitral stenosis; though Osler states that such women pass through repeated pregnancies with safety, and he places stenosis in women almost on a level with regurgitation. This view will hardly be taken by those who have witnessed appallingly sudden death occurring during pregnancy in the subjects of mitral stenosis, independent of the special dangers attending the process of delivery.

The average age of all patients dying from mitral stenosis probably does not exceed thirty-three years: Broadbent gives the figures as thirty-three years for males and thirty-seven to thirty-eight for females. Sansom formulates the general rule that mitral stenosis discovered in young subjects rarely permits the extension of life to the age of forty years. Certainly the great majority of such cases succumb at a comparatively early age. Many authorities maintain that the average duration of life after the appearance of stenosis does not exceed ten or twelve years.

TRICUSPID REGURGITATION

Etiology.—A considerable degree of regurgitation at this orifice may be regarded as physiological, since it always occurs upon violent muscular exertion and is nature's provision for the relief of the temporary embarrassment caused by the distension of the right ventricle.

Rare as a primary pathological condition, it is nevertheless by far the most common form of valvular disease affecting the right heart.

Rheumatic endocarditis is answerable for a small proportion of the cases, but recent rheumatic tricuspid valvular disease, without any lesion in the aortic or mitral valves, is so rare that its existence may well be doubted.

Ulcerative or malignant endocarditis undoubtedly does affect the tricuspid valve in some cases where all the other valves escape.

Congenital disease of the valve is rare, and when present is usually associated with pulmonary stenosis ; it is probably the result of endocarditis in fœtal life.

The condition of *relative* insufficiency is a common event in left-heart disease, being produced by the dilatation of the right ventricle caused by the backward pressure of the blood in the lung in mitral and aortic disease, especially in the former, in which it occurs early. It is found in all conditions of the lungs, causing embarrassment in the pulmonary circulation, as emphysema, with bronchitis, cirrhosis, and bronchiectasis. Degenerative changes in the heart, the result of fibroid and fatty disease, and any marked loss of muscular power in the heart, as in pneumonia, fevers, diphtheria, &c., will produce relative insufficiency at the tricuspid. Babcock classifies the causes of tricuspid regurgitation as three : (1) Structural, (2) Relative, and (3) Muscular.

Symptoms.—These are such as have already been described as occurring in the failing compensation of mitral disease—cardiac oppression, dyspnœa which early becomes orthopnœa, venous engorgement producing cyanosis, pulmonary congestion, and hæmoptysis, hepatic enlargement, general anasarca, and ascites.

Physical Signs.—Inspection, palpation, and percussion will afford evidence of the primary lesion of the mitral or aortic valves by the secondary changes which they have induced in the heart, as enlargement, thrills, alteration of apex-beat, &c. In the same way the signs of the primary pulmonary lesions are evident.

The signs specially indicative of tricuspid regurgitation from whatever cause are—

1. Increased dulness to the right of the sternum, indicating dilatation of the right ventricle ; with this is associated visible

pulsation, which also extends to the epigastric and ensiform regions.

2. Visible pulsation and distension of the veins in the neck are seen. The pulsation is systolic, and is most marked in the right internal jugular, from the yielding of its valves owing to distension of the bulb. The veins, when emptied by the pressure of the finger, are found to fill from below. This systolic pulsation must not be confused with that transmitted from an underlying artery, which stops when pressure is applied to the vessel nearer the heart. If an auriculo-systolic venous pulsation is sometimes visible preceding the systolic wave, it is caused by the hypertrophied right auricle. But presystolic venous pulsation must in all cases be discriminated from the positive systolic venous pulsation, which is the only pathognomonic sign of tricuspid regurgitation.

When the muscle-power is failing in the right ventricle there may be no evidence of pulsation in the veins, but when the opposite condition prevails the systolic pulse may be seen in the axillary and other veins.

3. Hepatic pulsation may be obvious, if the lower part of the swollen organ be grasped between the hands; in this way the regurgitant wave transmitted through the inferior vena cava to the hepatic veins may be discriminated from transmitted aortic or cardiac pulsation.

4. The chief auscultatory sign is a murmur of soft blowing character, whose point of maximum intensity is over the fourth or fifth space immediately to the left of the sternum, and whose direction of conduction is sometimes towards the right axilla; it may often be heard also at the apex, but never at the back. It is systolic in rhythm and is obviously caused by regurgitation of blood into the auricle during the systole of the right ventricle, and it becomes very faint on deep inspiration. This murmur may be heard almost anywhere outside the tricuspid area, and hence its significance must be always doubtful if venous pulsation be absent.

5. Pitt points out that in mitral stenosis, as the second pulmonary sound is always accentuated, it is a valuable sign of the supervention of tricuspid leakage should this sound become markedly feeble.

Diagnosis.—The soft systolic murmur inaudible at the back, with systolic venous pulsation in the neck and liver, and feebleness of the second pulmonary sound, are characteristic. The increase in the size of the jugulars, which in some cases become enormously distended, cannot be overlooked. In one case of tricuspid regurgitation recently seen by the writer to occur in double pneumonia in an aged lady, the neck-veins above the right clavicle became distended, forming a remarkable pulsating tumour of the size at least of a small goose-egg.

Prognosis.—This, except in acute cases the result of transient strain, is always most grave ; in all other cases the prognosis is that of the original lesion which, in its last stages of failure of compensation, has led to the regurgitation. It is a very rare event to see compensation even partially restored after the supervention of tricuspid leakage in mitral or aortic disease.

TRICUSPID OBSTRUCTION

Etiology.—This rare condition is sometimes congenital. When found after infancy it is almost invariably of rheumatic origin, and nearly always is secondary to mitral stenosis, as proved by Pitt's figures. Females are much more frequently affected than males, and the most common period is between late childhood and middle age.

Symptoms.—The symptomatology of this valvular affection is often very obscure. Since, if it existed alone, there would be no lung-symptoms, it must therefore be obvious that the clinical picture is that of the associated lesions when the lungs are affected. Lividity or extreme cyanosis, with distension of the jugulars and general venous engorgement and great œdema, preceded by hepatic enlargement and albuminuria, are obvious features which contrast with those seen in mitral stenosis, unless in the very last stages of that lesion.

Physical Signs.—These are the signs of the primary mitral stenosis, to which the tricuspid obstruction may be regarded as always secondary. The condition is seldom diagnosed during life, owing to the frequent absence of the most characteristic sign, which is a presystolic murmur, heard, however, in only about 12 per cent. of all cases. Its site is at the tricuspid area ; its quality is harsh or high-pitched ; its frequent absence is probably owing to the small proportion of muscle natural to the right auricle, which seldom is capable of taking on a fair degree of hypertrophy. A systolic high-pitched murmur is present in a much larger proportion of cases ; it is audible over the right ventricle in the ensiform and epigastric region, and is obviously the result of the tricuspid regurgitation which is usually also present. A presystolic thrill just below or between it and the lower left costal cartilages may be also present.

Diagnosis.—As stated, this is difficult and often impossible, owing to the uncertainty of the distinguishing characters of the mitral presystolic as contrasted with the tricuspid presystolic murmur. Where the condition is suspected, the early supervention of extensive dropsy is in favour of the existence of tricuspid stenosis.

Prognosis need not be discussed in a condition about whose diagnosis so much uncertainty exists, especially as the

outlook is largely dominated by the extent of the primary mitral obstruction; and when the symptoms arouse the suspicion of secondary tricuspid stenosis the case is always beyond hope of even temporary improvement being possible.

PULMONARY REGURGITATION

This is the rarest of all valvular lesions.

Etiology.—When the disease is due to congenital malformation the number of cusps may be increased, or three of them may be fused into two during a fœtal endocarditis, and some stenosis is usually present. Ulcerative endocarditis is the cause of the majority of the short list of recorded cases. Dilatation of the pulmonary artery and its ring, as in the cases of aortic regurgitation, caused by dilatation of the aorta, produces *relative* insufficiency in the healthy valves guarding the artery. The cause of this dilatation is long-standing disease of the left heart, especially mitral stenosis. Aneurism of the aorta pressing on the trunk of the pulmonary artery near to its orifice acts in the same way.

Symptoms.—These are produced by the primary lesion in the first instance, and as in most cases of the structural types this is malignant ulceration the clinical features are those found in ulcerative endocarditis. In the cases caused by relative insufficiency, the symptoms are those of the primary mitral disease or other valvular affection.

At a late stage in all cases, the usual venous engorgement, as seen in cyanosis, dyspnœa, dropsy, pulmonary hæmorrhage, or infarctions, &c., is seen. In most cases the symptoms are entirely latent till pulmonary embolism or failing compensation appears.

Physical Signs.—There is evidence of great enlargement of the right ventricle, in the displacement of the strong heaving apex-beat and extension of the cardiac dulness beyond the right margin of the sternum. Sometimes a systolic thrill is present; but in the main the signs are those of the primary valve-lesion in the relative cases, and in the structural or organic cases there is usually little to suggest the condition till the right ventricle dilates.

A soft diastolic murmur is present with maximum intensity at the third left intercostal space, and conducted along the left margin of the sternum downwards. It is not heard in the neck or to the right of the sternum. It is rendered more intense during prolonged expiration and by the patient assuming the erect posture.

Diagnosis.—This diastolic bruit so closely simulates that of *aortic regurgitation*, which may sometimes be heard at the same site, that a diagnosis is difficult or impossible. If the main bulk of the hypertrophy be clearly identified with the right and not with the left ventricle, and if the aortic second

sound be clear and the pulmonary indistinct, the presumption is that the murmur is produced at the pulmonary valves. The most valuable aid is obtained by a careful examination of the arteries and radial pulse. The water-hammer or Corrigan's pulse, the presence of visible pulsation in the arteries, the phenomena of venous and capillary pulsation, and Duroziez's sign, all point conclusively to aortic regurgitation. When both lesions occur together, the murmurs are indistinguishable. Barié lays great stress upon the importance of the feebleness of the pulmonary second sound.

Prognosis.—In relative insufficiency, the primary lesions being generally of a fatal nature, as malignant endocarditis, aneurism, and aortic or mitral disease, the outlook is always grave, especially as the pulmonary valves do not show regurgitation till the late stages of these affections. In primary or structural cases, where the lesion is not associated with other valve-disease, the duration of life may be little shortened.

PULMONARY OBSTRUCTION

Etiology.—Stenosis of the pulmonary orifice is almost always a congenital malformation, and is the most common of these. It is, however, rare as an isolated malformation, being usually associated with open foramen ovale and other deformities, as described upon page 583.

As an acquired or post-natal rheumatic affection, it is one of the rarest of conditions. It has been known to result from atheroma of the pulmonary artery, from the pressure of an aortic aneurism, and from ulcerative endocarditis, in which latter case the tricuspid is also usually found stenosed.

Symptoms.—These are such as have been described under cardiac malformations (see page 584), the chief feature being cyanosis and dyspnœa on exertion. In the acquired form of the disease pallor is more commonly present than cyanosis.

Physical Signs.—These are very variable and uncertain. A considerable degree of bulging at the lower end of the sternum caused by hypertrophy and dilatation of the right ventricle is always present. The most characteristic sign is the presence of a murmur, blowing and rough or harsh in quality, of varying duration and loudness ; its site of maximum intensity is at the left base in the third interspace, and it may be heard all over the chest, but is not obviously conducted along the vessels in the right side of the neck ; it is systolic in rhythm. A fine systolic thrill is felt above its site of maximum intensity. A diastolic murmur, indicative of pulmonary regurgitation, may accompany the weakened or absent pulmonary second sound ; when this murmur is absent the sound may be accentuated.

Diagnosis.—The chief difficulty lies in the exclusion of *aortic stenosis*, as the murmur closely simulates the bruit

produced at the narrowed aortic aperture; its line of propagation is, however, along the branches of the pulmonary artery, and not in the direction of the neck-vessels. Another sign of diagnostic value is that the chief hypertrophy and dilatation belong to the right and not to the left ventricle. The sphygmogram of aortic stenosis is a valuable aid.

A pulmonary murmur is extremely common without any disease of the valves, as observed in anæmia and chlorosis, constituting the systolic hæmic murmur, which disappears after a course of iron. Cardio-respiratory murmurs (page 610) are sometimes heard in the pulmonary area, and a well-marked, loud, and rough systolic bruit is often heard in thin-chested young muscular subjects, which disappears entirely whilst the breath is suspended after a deep inspiration. Broadbent explains this condition by the absence of the thin edge of the overlapping lung permitting the flattening of a part of the conus arteriosus of the pulmonary artery against the chest-wall, which produces an eddy in the current of inrushing blood in the artery.

COMBINATIONS OF VALVULAR DISEASE

These have been incidentally referred to in the descriptions of the individual valve-lesions. Of the isolated valve-affections, as already stated, mitral regurgitation is by far the most common, and, though often associated with mitral stenosis, nevertheless the combination most frequently met with in practice is probably mitral regurgitation with aortic regurgitation. If, however, we include the tricuspid relative insufficiency which so often appears in the late stage of the mitral affection, this combination is the most common; then will follow next in frequency the triple association of mitral, aortic, and tricuspid regurgitations.

All sorts of combinations may be met with, and it is not unusual to hear the double murmur of aortic stenosis and insufficiency in the same case where the mitral area furnishes the double murmur of mitral obstruction and regurgitation. Mitral stenosis and aortic regurgitation, or mitral stenosis and aortic stenosis, may occur together; these combinations are, however, rare. Walshe gives the most frequent twin combination as that of aortic stenosis and mitral regurgitation; but this is certainly as incorrect as his statement that aortic stenosis stands second in the list of all single lesions after mitral regurgitation.

The diagnosis usually does not cause much difficulty if the sites and rhythms of the murmurs are carefully investigated and an analysis of the symptoms in failing compensation be made—points already discussed under each separate lesion.

The *prognosis* in the combinations of valve-disease is that already discussed under the heading of each isolated form.

It is needless to say that, speaking generally, the prognosis is more serious in proportion to the number of the valve-lesions present in any given case. In the forecast of combined mitral lesions an important statement made by Osler should be borne in mind. This authority, speaking about the prognosis in mitral regurgitation, states that 'the cases which last the longest are those in which the valve-orifice is more or less narrowed as well as incompetent.' This is in harmony with the clinical experience of most physicians, as these lesions are usually regarded as antagonistic to each other. The prognosis in the combination of mitral regurgitation and aortic stenosis is, upon the other hand, always most grave, as it is obvious that the aortic stenosis intensifies the regurgitation. Aortic regurgitation, when combined with mitral regurgitation, has not so grave a prognosis as might be expected, provided both lesions are primary ; but if the mitral lesion is relative or secondary it indicates extreme gravity, inasmuch as it is proof of dilatation and muscular failure. Aortic stenosis, if of mild degree, rather improves the prognosis of aortic regurgitation, since, as Babcock suggests, it may serve to protect the ventricle against the too free influx of blood.

TREATMENT OF CHRONIC VALVULAR DISEASE

In the author's 'Dictionary of Treatment' (4th ed.), the treatment of valvular disease is dealt with in detail upon pages 389–405 ; only the general principles need be here mentioned.

In those cases where the presence of a murmur during an attack of acute rheumatism shows that one of the cardiac valves has become involved, it becomes a question of vital interest to the patient whether anything can be done to prevent the permanent establishment of stenosis or insufficiency at the affected orifice. There cannot be a doubt that a prolonged course of absolute rest for several weeks, or even months, after the cessation of all rheumatic symptoms will markedly diminish the chance of a permanently crippled condition of the valve. This will be again referred to under the heading of 'Rheumatism.'

Where in an apparently healthy individual a valvular lesion is discovered in a routine examination, though drug-treatment is entirely out of the question, it becomes a matter of serious importance as to how far any other interference is permissible. With a knowledge of the peculiarities of the individual's temperament and habits, the physician will judge for himself how far he is justifiable in taking him into his confidence and explaining the condition of affairs. Where there is no obstacle or objection to such a course, the subject of a valvular lesion in the stage of complete

or perfect compensation should receive instructions as to the regulation of his daily life. This is a matter of no difficulty, since such instructions for the greater part are but a repetition of the ordinary laws of health.

Exercise is the only point about which there is a likelihood of mistakes being made: not only should exercise be not forbidden, but a considerable amount of muscular exertion should be insisted upon. Running for long or short distances, football, boat-racing, weight-throwing, and athletic training are to be condemned; but most other exercises are essential, and none so good as walking, which may be freely indulged in; even mountain-walking, when done leisurely, is a valuable agent in keeping up the tone of a crippled heart. No form of exercise, however, should be pushed to the extent of producing cardiac discomfort, and in aortic insufficiency this is the more liable to be induced. The subject of such a valvular disease may be generally assured that, though he may steadily walk a distance of many miles with advantage, the short spurt of running a hundred yards to catch a train may be accompanied with grave danger.

Occupations involving severe muscular strain, and others which are associated with much mental worry and excitement, should be given up.

Diet should be such as the patient's previous experience has demonstrated to be suitable. Tobacco and alcohol must be used sparingly, and tea in large quantities should be forbidden. Regularity in meals, and in the alternating periods of work, rest, and sleep, should be insisted upon; late suppers are to be condemned, especially after long fasting, and sexual excesses are fraught with danger.

Drugs in the stage of compensation are inadmissible: the cardiac tonics especially, in the absence of all symptoms, are prone to do harm; but iron for anæmia and the vegetable laxatives, when constipation is present, are clearly indicated.

Exposure to extremes of cold and heat should be guarded against, and the Turkish bath is to be avoided. The subject of mitral regurgitation can usually be assured that, by temperate living and attention to details like those already mentioned, his heart-affection is compatible with a long life.

Treatment of Failure of Compensation.—When symptoms of failure in compensation show themselves, they generally first appear after some unusual degree of exercise, or follow the debility produced by intercurrent affections, and they demand immediate attention.

Rest as absolute as possible should be the first essential in all cases; breathlessness, slight œdema, palpitation, cough, and even cyanosis may often be found to yield to enforced rest in the horizontal position without the use of any drug. The benefits of this agent are in direct proportion to the

thoroughness with which it is carried out, and much depends upon the character or temperament of the patient. Some victims, in whom compensation is evidently failing, refuse to take to bed in the daytime, and struggle upon their feet against dyspnœa till finally they become unable to lie down even at night. Others will rest quiet in bed for days, but will insist upon suddenly getting up and walking to the water-closet. Rest under such circumstances may be positively injurious. A patient with a dilating heart, after several days' complete rest, upon suddenly assuming the erect posture, places himself in a position of greater danger than if he had continued to move about his room daily from the beginning of his treatment. Others, it must always be remembered, are really unable to lie in the horizontal position, and are compelled to sit up day and night in an arm-chair. The physician must therefore study the individual before enforcing the cast-iron edict of absolute rest in the horizontal position. There is another detail in connection with this subject which may lead to disaster, unless the physician be careful. With some patients the use of the bed-pan is an impossibility, and the amount of straining which the attempt occasionally produces may induce a fatal issue ; though rest is so very beneficial and desirable, it is nevertheless imperative in some cases that the patient be assisted gently and deliberately to the night-chair placed beside his bed. In all cases, however, he should be made to pass urine in the horizontal position when his symptoms are urgent, and constipation should be prevented by every means.

Diet during failing compensation must be carefully attended to, but each case must be considered on its merits. As a rule, the patient's own inclinations should be studied ; it is a mistake to insist upon the rule of enforcing a rigid dietary. Some patients do well upon a dry diet ; others can only manage to exist upon liquids.

A common error is the routine employment of excessive carbo-hydrates, and Steell rightly insists upon the reduction of these to a minimum, and the administration of nitrogenous food upon the lines pursued in ordinary wise living. Three meals may be given daily, breakfast consisting of a large cup of boiled milk flavoured with tea, and accompanied by a boiled or poached egg and cold buttered toast. The mid-day meal may consist of chop, fish, or fowl, with green vegetables and fruit. He permits a cup of afternoon China tea without any bread or biscuit. The evening meal is upon the same scale as the midday one.

An absolute milk-diet is only recommended by the above-mentioned authority when the patient is wholly confined to bed and is suffering from the complication of renal disease and high tension in the vessels. The value of rectal feeding

is obvious where gastric catarrh and dyspepsia are prominent symptoms, and various formulæ for the preparation of nutrient enemata will be found in the article in the author's 'Dictionary of Treatment' previously referred to.

Cardiac Tonics.—As already stated, these are always contra-indicated in the stage of compensation, and should never be administered till symptoms of failing compensation have shown themselves. Digitalis stands at the head of the list as the most reliable of all drugs which exert a true tonic action upon the cardiac muscular fibre. Though it undoubtedly exercises its power also upon the cardiac ganglia, vagus-roots, and terminals, and upon the capillary walls, it must always be remembered that the main effect of the drug in medicinal doses is upon the muscular fibre of the heart. The chief, and it may be said the only, indication for the administration of digitalis in chronic valvular disease is the presence of symptoms indicative of *failing muscular power* in the walls of the heart, independent of the site or nature of the valve-lesion. The drug may be regarded in one sense almost as a cardiac food, since the muscular fibres increase in size and number under its administration. It may be given in ruptured compensation in every form of valvular disease, though some authorities still hold that it is contra-indicated 'in the early stages of aortic insufficiency'; but it must not be forgotten that it is contra-indicated in the early stage of all valvular lesions. Undoubtedly it acts most beneficially in aortic disease when the hypertrophied ventricle has commenced to dilate, and when relative insufficiency of the mitral has supervened. Its power of prolonging life under such circumstances is an undisputed fact.

The labours of Gaskell and others, referred to in the description of heart-block upon page 568, have modified many of the views formerly held regarding the action of the digitalis group. Dr. John Hay, in an able paper appearing in the 'Practitioner' of October 1907, sums up the chief results of these investigations and of the researches of Mackenzie and Wenckebach as proving that each cardiac muscular fibre possesses five inherent qualities, though certain groups of fibres are more highly endowed with one function than another. Thus he states that, as proved by Gaskell, 'the heart-muscle possesses in itself the power to originate its own stimulus to contraction; that the cardiac muscular fibres are able to conduct that stimulus from one part of the heart to another; that these fibres are excitable, and can respond by contracting in response to the stimulus when it arrives; and that the myocardium possesses the important characteristic of tonicity.'

Digitalis is therefore to be accepted as acting upon the tonicity of the heart-muscle, improving it; also upon its con-

tractility, augmenting the force of the systole ; and, furthermore, upon the frequency of the beats by depressing the function of stimulus-production or the excitability of the musculature, either by direct action on the muscular fibre or by influencing the vagus-terminals in the heart.

In mitral insufficiency with dropsy its best results are obtainable, but in stenosis of this valve, owing to the smaller mass of muscle in the enlarged left auricle, its action is correspondingly limited ; when, however, the right ventricle has become weakened and is beginning to dilate its influence for good is often very striking, the signs of pulmonary embarrassment sometimes speedily giving way.

Not only is the diastole lengthened, during which period the heart is rested and fed, but the relative sizes of the mitral and tricuspid orifices are lessened, so that the valves are aided in their function. Its action upon the arterioles must always be remembered, for by increasing peripheral resistance it raises the general and renal blood-pressure, and to this is attributed its striking value when dropsy has occurred. Where the arterial tension is already high, as in valvular disease associated with Bright's disease, it may be necessary to administer a vaso-dilator like nitroglycerin in combination with it ; or strophanthus may be used instead of digitalis, since it has less power in constricting the small vessels and less influence over the vaso-motor centre.

When possible at the beginning of a course of digitalis-treatment rest should be enforced ; often the drug fails to relieve the symptoms till the patient takes to bed. The cumulative action of digitalis figures largely in the writings of pharmacologists, but at the bedside it is very seldom witnessed ; it should never be met with, since this result cannot occur unless the urinary secretion becomes markedly diminished. Hence in a course of active treatment by this drug the daily amount of urine should be measured, and upon any marked diminution in the quantity the administration should be suspended or the dose diminished for a time. The increase in the urinary secretion is always marked in dropsical cases, and gives a fair indication of the benefits to be expected from the drug in a given case after sixty hours from the beginning of the treatment. The pulse becomes slower, fuller, and more regular, and the tension markedly improves. Increase in frequency, or very pronounced infrequency, of the pulse, or the condition in which the heart beats twice for once that the pulse is felt in the radial, or the supervention of much irregularity, are indications that the drug should be discontinued or the dose diminished. It is in the last-mentioned state of the pulse that syncope is likely to occur, and hence it is advisable to warn the patient

against suddenly sitting up in bed when full doses are being administered in cases of great cardiac failure.

Dosage is a matter requiring much skill and experience. As a rule, when the patient consents to begin his treatment by rest in bed, a moderate dose—say, 10 mins. of the tincture thrice daily—is usually sufficient. When the urgency of the symptoms demand large doses it will be well to remember that, owing to the length of time required for the full action of the drug, it may be necessary to start with a full hypodermic dose of strychnine. In extensive dropsy larger amounts of digitalis are indicated, and are usually tolerated without difficulty and without danger. It is well in severe cases to give the drug in the form of tincture or infusion without any adjuvant, so that the tolerance of the stomach can be properly tested. Little importance need be attached to the contradictory statements about the relative values of the different preparations. The powdered leaf in doses of 2 grs. (equivalent to 16 mins. of the tincture) may be given in the pilular form, and this amount seldom needs to be increased. Nativelle's crystalline digitaline in granules, each containing $\frac{1}{240}$ gr., is an elegant and convenient form, but inferior in results to the leaf or tincture. Whatever preparation is employed, there should be a sufficient interval between the doses to admit of elimination, and when possible eight hours should elapse between each full dose after the drug has been given at short intervals for two or three days.

The experience of every physician affords instances in which digitalis has been thus continuously employed for periods of many weeks or months ; but, upon the whole, it is wiser to suspend the drug for two or three days occasionally, even when the urinary secretion is copious. Usually, after free diuresis has been established, by the end of the fourth day after commencing treatment, the dose may be diminished, or the interval between the doses extended to twelve hours. In some cases free diuresis only occurs after the use of the drug has been suspended for about three days ; when such result occurs the digitalis should be at once resumed in smaller doses given at lengthened intervals.

Strophanthus stands next to digitalis in value, and in a small proportion of cases it succeeds after the failure of the older remedy. In the writer's experience it is more trustworthy in pure mitral stenosis where the left ventricle is not hypertrophied—a condition in which digitalis is liable to fail and be ill-tolerated. It is certainly inferior in diuretic power, though it often removes the marked irregularity of the pulse. The 'B.P.' dosage of its tincture is identical with that of digitalis ; but Haynes has shown that the tincture of strophanthus is ten times more toxic. In the present state of our knowledge of the complex pharmacological actions of these

two drugs it is unwise to combine them ; though there can be no objection to alternating courses, provided three days at least be allowed to intervene between the cessation of one course and the beginning of the next.

J. Hay states that a case of mitral stenosis in the first stage, with a normal manner of systole and a frequent action of the heart, generally fails entirely to slow down under digitalis and its allies ; but when there is evidence of failing tonicity or ineffectual ventricular power the drug is serviceable, irrespective of its effect upon the pulse-rate.

Strychnine possesses virtues which in some respects place it above digitalis and its rival in the dilatation of ruptured compensation. Its main value lies in the rapidity of its action, but to ensure its effects it must be given hypodermically. For continuous or long-sustained effect it is much inferior, but great benefits may be obtained by combining it with digitalis or strophanthus when a prolonged course of cardiac tonic treatment is indicated.

The relative values of sparteine, caffeine, lily-of-the-valley, adonis vernalis, and the other drugs recommended as cardiac tonics in failing compensation, will be found in the author's article already referred to.

After compensation has been restored by rest and digitalis, and the symptoms produced by dilatation have subsided, the greatest care and discretion should be observed in permitting the patient to move about or take exercise, otherwise the weakened ventricle will again rapidly dilate.

Symptoms requiring special treatment usually present themselves in every case of severe ruptured compensation, since the action of the cardiac tonics is too slow to meet the urgency of such embarrassments as may arise from extensive dropsy or portal and pulmonary congestion. Thus purgatives of the saline group are generally indicated to produce free watery evacuations, in order to remove serous effusions and assist the overburdened kidneys and liver. Hay's method of administering concentrated solution of magnesium sulphate often produces striking results. Calomel has its strong advocates, and the brilliant successes achieved by the judicious administration of mercurials by the former generation of practitioners, who used blue-pill extensively in the treatment of failing compensation, is liable to be forgotten. The old formula of Guy and Baly, consisting of 1 gr. each of blue-pill, squill, and digitalis, owes much of its value to the metallic element, and often succeeds after all other combinations have failed.

Diuretics like caffeine citrate, diuretin, and nitroglycerin are indicated when the urine ceases to be increased by digitalis.

The pulmonary and general venous stagnation may urgently call for a rapid diminution of the volume of blood without

waiting for the action of purgatives, and this is imperative in acute dilatation, when life can only be saved by venesection, one or two pounds of blood being removed from an incision in a large vein.

Dyspnœa often urgently demands relief, and Allbutt has demonstrated the great value of morphia hypodermically. It should be given with atropine, and may be administered even when there is marked albuminuria, should the distress be acute.

Insomnia is sometimes so terrible in the exhaustion which it produces, that at all hazards sleep must sometimes be induced by hypnotics. The most certain and reliable is morphia hypodermically; but this agent in full hypnotic dose is fraught with considerable danger, especially when a dropsical state of the lungs is present. Paraldehyde is absolutely safe when the patient can be induced to swallow a full dose of 100 mins. mixed with whisky or wine. Chloral should never be given, and sulphonal usually intensifies the misery by producing a state of prolonged drowsiness which does not end in sleep. When paraldehyde fails singly, it may be combined with a moderate dose of morphia.

Such symptoms as cough, hæmoptysis, flatulence, diarrhœa, and vomiting are to be treated upon general principles, always, however, remembering that they are but local manifestations of the primary lesion which has mechanically produced the venous stasis to which these symptoms owe their origin.

The very frequent association of anæmia with valvular disease must not be overlooked, and the combination of iron with digitalis is indicated at some stage of nearly every case.

The value of iodides in the relief of cardiac pain, especially in aortic disease, is often most striking, and the indication of nitrites for the relief of angina-like paroxysms is obvious.

The value of the treatment of Oertel, and that of Schott, as carried out at Nauheim, is discussed in the author's article already referred to.

HEMIPLEGIA

This is the name given to the motor paralysis which affects one side of the body, with or without impairment of sensation. By the term 'one side of the body' is usually meant the lower part of the face, the tongue, the leg, and the arm.

The lesion is caused by disease or injury to the motor centres or the motor path in some part of its course from the cortex of the central convolutions through the centrum ovale, internal capsule, crusta of the peduncle of the cerebrum, the pons, and anterior pyramid of the medulla. As the paths cross each other or decussate in the medulla, left-sided hemi-

plegia implies involvement of the right motor tract, and *vice versâ*.

The common site of the lesion is at the posterior limb of the internal capsule, where the converging fibres on their way to the opposite side of the body are all represented.

The most frequently occurring cause of hemiplegia is hæmorrhage into this region, and the symptoms and varieties of the affection are described under the heading of 'Cerebral Hæmorrhage' (page 177), where also are mentioned the other lesions which produce hemiplegia.

HERPES

This affection, also known as Zona, Shingles, or Herpes Zoster, is characterised by the presence of an acute vesicular eruption following the lines of distribution of the superficial nerve-fibres proceeding from the ganglia on the posterior roots.

Etiology. — The disease occurs at all ages, but it is most common in childhood, about three out of every four cases, according to Head, being found in subjects under twenty-five years, and the largest number of these occurring between four and thirteen.

Age influences the severity of the affection, the eruption in subjects of advanced age being generally associated with great pain. Sex has little influence. A small number of cases are symptomatic or secondary to some disease of the nerves, as in ataxia, myelitis, cerebro-spinal and tubercular meningitis, and general paralysis of the insane; others are associated with spinal caries and necrosis of the ribs; and in a few instances the eruption has followed direct injury to superficial nerves.

Hutchinson pointed out the influence of arsenic, when taken for therapeutic purposes, in producing true herpes, and it has been known to follow the inhalation of carbon monoxide and other poisonous gases. Dr. Eustace, of Widnes, informs the writer that he has had under his observation several cases of *bilateral* herpes produced by the inhalation of arsenic-laden dust, and he regards rightly the bilateral distribution of the eruption as proof of its toxic origin in all cases.

Mental emotion and exposure to severe cold and chills are regarded as etiological factors. In by far the larger number of cases there is no apparent cause, the disease occurring sometimes in epidemic form, like one of the specific fevers.

Symptoms.—There is usually a feeling of malaise and some febrile disturbance, which may continue for three or four days before the appearance of the eruption. But the most constant symptom is a smart neuralgic pain in the region afterwards occupied by the eruption. This pain may precede

the first crop of vesicles by a few hours, or it may be slight or severe for several days before the appearance of any rash.

The eruption consists of vesicles, or papules which rapidly develop into vesicles, irregularly studded in groups or clusters over an erythematous base. In some cases the erythema may be observed before the appearance of either papules or vesicles. The contents of the vesicles are at first clear, but they soon become turbid, and occasionally purulent or blood-stained. They vary in size from that of a hemp-seed to that of the section of a large pea, and sometimes numbers of them coalesce, forming large bloody bullæ. The eruption usually develops rapidly, and reaches its maximum in two or three days, though sometimes successive crops of vesicles may continue to appear for six or seven days, after which the vesicles shrivel, dry up, and form scabs, under which by about the end of a week or later the skin is found completely healed, only a slight scarring remaining. In severe cases the scarring may be deep and permanent, causing considerable disfigurement.

The pain is generally not markedly relieved by the appearance of the rash: it may be even exaggerated then; it is usually associated with considerable itching, and it is liable to exacerbations at night. As a rule, it disappears after the scabbing, but in old subjects it may last for long periods as a severe burning neuralgia, and in some cases, fortunately rare, it may never disappear when the scarring has been deep and extensive. More or less hyperæsthesia precedes and accompanies the development of the rash, and often a considerable degree of anæsthesia remains where the scarring has been deep.

Distribution.—The site of the eruption is characteristic. As already stated, it corresponds to the distribution of the superficial nerve-fibres arising from a posterior root-ganglion, and in the trunk it forms a horizontal band which encircles one side of the body like a girdle. Occasionally the vesicles may be seen extending to a very limited extent beyond the middle line behind and in front. This slight overlapping, however, does not influence the almost invariable rule that the eruption is *unilateral*. In the extremely rare cases where it has been bilateral the two half-girdles have been at slightly different levels.

The distribution does not correspond to a single nerve-area, but affects the region within the posterior root-ganglion zone of the dorsal, cervical, or lumbar nerve involved. By not strictly following the oblique direction of any special intercostal nerve, the band of eruption is horizontal on the trunk. The continuity of the girdle is usually more or less broken, the clusters of vesicles being thickest over the regions where the nerve-fibres emerge from the fascia—viz. near the

spine behind, near to the middle line in front, and about the longitude of the axilla in the middle.

Upon a limb the eruption follows the course of the nerve-fibres, and various names have been invented to designate the regions involved, as H. brachialis, cruralis, intercosto-humeralis, &c. Any region may be involved, but the eruption is rare on the foot and lower part of the leg.

One formidable variety of great importance, on account of the injury and suffering entailed by it, is when the upper part of the face is affected. Here the Gasserian ganglion is involved, and the distribution of the eruption may correspond to the entire area supplied by the ophthalmic division of the fifth nerve—viz. the forehead and scalp as far back as the parietal prominence, and in front involving the eyelids and side of the nose. This H. ophthalmicus is associated with intense pain ; ulceration of the cornea and iritis often occur, and even ophthalmoplegia and blindness have followed. Sometimes severe and intractable neuralgia remains after the healing, and in one case recently seen by the writer some sloughing of the frontal skin followed, and obstinate ulceration remained for a considerable period. Scarring always follows.

When the occipital nerve is involved the eruption invades the scalp, and has been followed by persistent neuralgia.

One noteworthy feature of true herpes zoster, on a par with its unilateral distribution, is the extreme rarity of a second attack, in which peculiarity it resembles the exanthemata. It is known also to have a seasonal prevalence, or to appear in small epidemics more or less under the influence of obscure atmospheric conditions.

Pathology.—Herpes zoster is to be regarded as an acute specific disease of the nervous system. According to Head, it is analogous to poliomyelitis anterior acuta. The cause is probably a toxin of microbic origin, which produces an acute hæmorrhagic inflammation of the ganglia on the posterior nerve-roots, destroying their cells. The results of *post-mortem* examination have demonstrated this ; many observers have also found secondary degeneration in the posterior roots and in the peripheral sensory nerves in connection with the ganglion. These changes are so definite that Head and Campbell were able to trace the distribution of the degenerated posterior root-fibres in the posterior columns of the spinal cord.

The vesicles of the eruption have been found to have their origin, like those of eczema, in the upper part of the rete, but the protozoan bodies discovered in them by Pfeiffer are regarded by Unna and others to be simply altered epithelial cells. The nerve-twigs are the seat of interstitial inflammation. Changes identical with those found in the spinal ganglia have been observed in the Gasserian ganglion.

Herpes facialis, or *H. febrilis*, is a distinct affection from zoster, and is only here mentioned in contradistinction. It is the name given to the herpetic eruption so common on the lips, nose, and sometimes on the cheeks, and occasionally upon the pharynx, palate, and tongue, in epidemic catarrh, pneumonia, &c. It is usually a bilateral affection, and also, unlike zoster, it frequently recurs, and is not accompanied by neuralgic pain. Crocker regards this eruption as of neurotic origin, possibly a reflex irritation of the sympathetic ganglia of the affected region through the sensory nerves of the part. This he believes to be the explanation of *Herpes præputialis*, a vesicular eruption appearing upon the inner surface of the prepuce. This, however, cannot be the pathological condition of those inveterate cases of the affection which the writer has observed in ataxia, and which resist all treatment. Recently Howard has recorded the results of a careful examination of the Gasserian ganglion in a number of fatal cases of pneumonia where herpes was present during life, and found the ganglion always diseased upon the side where the vesicles were markedly present.

Herpes iris is totally distinct from zoster ; it is mentioned under ' Erythema.'

Diagnosis.—The prodromal symptoms, the presence of neuralgic pain, the unilateral distribution of the eruption following the course of a branch or branches of one of the cutaneous nerves, and the vesicular nature of the eruption itself, are all characteristic.

Prognosis.—The disease always tends towards speedy recovery, but a guarded opinion is necessary in relation to the amount of neuralgia and scarring which may remain. These are often in direct relationship to each other, and are prone to follow the healing process in the aged and in herpes ophthalmicus, in which latter condition serious conjunctivitis and iritis may supervene.

Treatment.—Free dusting over of the site of the eruption with zinc powder and starch, or boric acid, and a light layer of absorbent wool to protect the part from injury or friction, suffices in most cases. Ointments are better in case the vesicles have ruptured, and when the pain is excessive cocaine and a little menthol should be added. Severe neuralgia must be relieved by morphine hypodermically.

HICCOUGH

This can only be regarded as a *symptom* of various diseased conditions, though its importance demands a separate notice. It is produced by a sudden spasmodic contraction of the diaphragm before the glottis is sufficiently opened to receive

the incoming air. The phenomenon is produced, according to Poore, by the absence of the normal synchronism which characterises the descent of the diaphragm and the opening of the glottic aperture, the noise being caused without any spasm of the glottis, as is usually believed by physiologists, since it does not occur during the normal inspirations which separate the individual 'hiccups.'

Etiology.—The efferent impulse is conveyed to the diaphragm by the phrenic nerve, and any irritation direct or reflex may induce the symptom. The most common causes are the rapid swallowing of very hot or cold liquids, the accumulation of flatus, and the irritation induced by the products formed in dyspeptic conditions. It is a symptom of many inflammatory states of the abdominal organs, as gastritis, obstruction from hernia, bands, &c., and in every disorder which produces peritonitis ; hence it is often present in perforation, appendicitis, and typhoid ulceration. Sometimes it appears as a grave symptom in gastric cancer and dilatation of the stomach.

Centric causes, as meningitis, cerebral tumour, or abscess, may produce hiccough through irritation of the region in which the phrenic takes its origin. The severe and sometimes fatal toxic form which occasionally is seen in Bright's disease, gout, diabetes, and liver-disease may belong to this group, or it may be possibly a reflex from the kidney or poisoned stomach.

When met with without any obvious or apparent cause, it has been regarded as a true neurosis, and exalted to the position of a separate disease allied to epilepsy. It is, however, probable that most of such cases are hysterical in origin, since obstinate paroxysmal hiccough is often met with in patients suffering from neurasthenia and hysteria, and the phenomenon may be both induced and removed in some patients by a profound mental emotion.

Prognosis.—In the vast majority of cases the symptom is a transient one and of little or no importance, but it must not be forgotten that it occasionally causes death by exhaustion, and this independent of the gravity of the primary disorder, though the prognosis may usually be regarded as that of the condition or disorder of which it is a symptom.

Treatment.— In the majority of trivial cases evacuation of the stomach-contents speedily arrests the spasmodic action of the diaphragm. By taking a deep inspiration and suspending respiration for as long a time as possible the symptom often disappears. The curative effect of a fright is often demonstrated by schoolboys, who have discovered the value of a profound emotional stimulant in stopping the symptom. Where the condition has lasted for some time, treatment should be directed to the primary disorder when this is

obvious ; but in the majority of severe cases it must remain empiric, one sedative or stimulant after another being tried till relief be obtained. Amongst the agents likely to prove useful are counter-irritation by sinapisms or ice to the epigastrium, pressure on the phrenic in the neck, forcible traction on the tongue for a couple of minutes, the induction of sneezing, pressure upon the supra-orbital nerve, and galvanism of the phrenic.

Of drugs the best is morphia in small doses, combined with cocaine given at short intervals ; but when the suffering becomes serious a full hypodermic of morphia or chloroform-inhalation must be resorted to. Amyl-inhalations and pilocarpine hypodermically sometimes give good results.

HODGKIN'S DISEASE

This is an affection characterised by anæmia and the presence of tumours, caused by enlargement of the lymphatic glands, which become the seat of hypertrophic adenoid growths ; the spleen, kidneys, liver, and other organs being usually affected in a similar manner.

The disease has unfortunately been confused with other affections owing to the multiplicity of its synonyms, as it has been described by various authorities under the names of Pseudo-leukæmia, Lymphatic Anæmia, Lymphadenoma, Lympho-sarcoma, Splenic Anæmia, and Lymphadenia.

Etiology.—Most of the published cases of this comparatively rare disease have occurred in young males ; it is very rare after middle-life. That local infection by some unknown specific organism is the important etiological factor is probable by the observations of Trousseau, who pointed out the frequency with which the first appearance of the disease had been preceded by the irritation of carious teeth, otorrhœa, and nasal catarrh in those cases where the enlargement originated in the cervical group of lymphatic glands. The influence of tubercle, syphilis, and parturition in a few cases is readily explained upon the theory of diminished resistance.

Symptoms.—These vary with the site of the tumours and with the rapidity of the lymphatic growth. The superficial glands are usually affected before the deep ones, and in many instances the first appearance of the disease is observed in the lymphatics in one posterior triangle of the neck. These increase in size slowly, when they may be felt as of almost stony hardness ; or their growth may be rapid, in which case they are soft and elastic to the touch, and may be slightly tender. They remain separate, and by manipulation the individual glands may be made to glide over each other,

and the skin is also freely movable over them. At a later period the glands in any large group may sometimes become conglomerated into a massive lobulated tumour, and it is not unusual to find at the same stage of the disease the enlarged glands quite separate and distinct in another region of the body. There is no tendency for the growth to invade or infiltrate the surrounding tissues.

The enlargement may commence in any superficial group, but occasionally the first evidences of the disease may show themselves by the painful pressure-symptoms which deep glands exert upon nerves, blood-vessels, or neighbouring organs. Not unusually the cervical or sub-maxillary group continues steadily to increase in size, and invasion of the thoracic glands becomes evident by spasmodic cough, thoracic pain, or dyspnœa. In the abdomen the tumours may cause various symptoms by their pressure on the bowel, ureters, iliac veins, and solar plexus ; œdema of the legs and bronzing of the skin are not uncommon. Interference with the venæ cavæ causes much dropsy, disturbance of the circulation, and cyanosis.

In some cases the latent enlargements may first arouse suspicions of their presence by anæmia, feverishness, asthenia, or emaciation ; but, as a rule, these symptoms belong to the late stages of the disease, when the lymphatic growths are far advanced.

After the enlargement of the superficial glands has lasted some weeks, usually the spleen can be felt below the ribs, and sometimes the lymphoid growths near its surface can be palpated, but the enlargement is never so great as that met with in leukæmia. Though moderate increase in the size of the liver is common, hepatic symptoms are unusual ; but jaundice from pressure on the bile-duct may occur. Renal symptoms are usually absent. When the lymphoid tissue-growths occur in the tonsils and pharynx, deglutition may be interfered with. When the stomach is involved, there may be serious dyspeptic trouble, and the deposit under the mucous membrane of the bowel may cause hæmorrhagic diarrhœa.

Fever of an irregular type is present in all acute cases in young subjects, and in the majority of all cases, for varying periods during the progress of the disease, but the temperature rarely exceeds 102°.

The blood-condition is important: the anæmia is of the secondary type, and may be very severe, the number of red corpuscles falling to 1,250,000 in 1 c.mm. in rare cases, and numerous small red cells or microcytes are often present. Leucocytosis is sometimes observed, but it is never marked ; in the majority of cases there is no increase in the number of leucocytes, though there is usually a relative increase in the

proportion of lymphocytes—*i.e.* in the small and large mononuclear cells. As already mentioned, in the later stages of the disease the emaciation and asthenia become extreme ; cardiac failure and irregularity of the pulse are often prominent, and death occurs from exhaustion or from asphyxia caused by pressure on the trachea or vena cava.

The duration of the disease is very variable ; sometimes the growths remain confined to a particular group of glands, which may remain stationary for considerable periods before general enlargement sets in, after which event the progress is always rapid. Those cases where the disease first shows itself by a widely diffused lymphatic enlargement run a rapid course and may terminate life in six or eight weeks. Gowers found that in fifty cases thirty-three died within two years ; more than the half of these, however, succumbed within twelve months from the beginning of the symptoms. Occasionally the duration may extend to five years, but in a small percentage of cases recovery has certainly been observed to follow treatment. Sometimes in chronic cases there may be noticed a marked diminution in the size, or even a total disappearance, of the tumours for a time, and this latter event has been observed in fatal cases.

Pathological Anatomy.—The lymphatic enlargement found after death corresponds with the symptoms and signs of the disease seen during life. The cervical glands show changes in most marked degree, and usually on both sides, but much more obvious on the one side than on the other ; and this latter is true of the other groups affected, the corresponding groups being commonly involved to a certain extent symmetrically. The size of the individual glands varies up to that of a small egg, and a mass of conglomerate glands may approach in rare instances to the magnitude of a small fœtal head. Chains of enlarged glands are usually found to connect those in the neck with the thoracic and the axillary groups. In the abdomen the enlargement of the retroperitoneal and pelvic glands may be very great ; the mesenteric are generally but slightly affected.

The glands are of a greyish-white colour, and may be soft or firm. On section the soft ones yield a juice consisting chiefly of lymphocytes with some multinuclear cells ; whilst those glands which are indurated are found to contain much fibroid tissue with few cells. The fibrous reticulum in the softer glands shows the meshes of its network to be crowded with lymph-cells, which are greatly increased in number and packed together between the fine fibrils of the stroma ; some giant and eosinophile cells are also present. The changes in the spleen are more or less identical. The Malpighian corpuscles enlarged by the growth of lymphoid tissue are of the same structure as the hypertrophied lymphatic glands, the

network of their fibrous reticulum being packed with lymphocytes. These growths are often numerous, and sometimes each may reach the size of a small grape ; they cut like a piece of bacon-fat, and may be surrounded by masses of pigment or infarcts.

Similar lymphoid growths are found in the tonsils, back of the tongue, the liver, and kidney, and to a variable extent may involve the solitary or Peyer's patches in the intestine. The medulla of the long bones is often found altered, the marrow being replaced by lymphoid tissue.

Pathology.—The pathogeny of the disease is unknown ; by many it is regarded as the result of an infective process caused by the invasion of some micro-organism like the tubercle bacillus, whose toxin has the power of acting as a direct stimulant to the adenoid or lymphoid tissue throughout the body. Muir maintains that the result of this infective process is essentially different from tuberculosis, and that the tissue-change can be readily demonstrated to be also different from lymphosarcoma, which is a true tumour-formation in lymphatic glands.

Diagnosis.—*Tubercular adenitis* or scrofula can be excluded by the following characteristics : Scrofulous disease of the glands is never general in its distribution, and very seldom bilateral, remaining often confined to the group through which the tubercular bacilli have gained their first entrance, as in the cervical or mesenteric regions. The natural tendency is towards caseation, necrosis and suppuration, or calcification—results practically never observed in Hodgkin's disease. The glands in scrofula inflame early, and the immediately surrounding tissues become involved, causing the matting together of the capsules of contiguous tumours, and the skin is soon invaded and ulceration takes place—an event which never occurs in the disease under consideration ; moreover, it is rare for tubercular glands to reach the degree of enlargement commonly observed in Hodgkin's disease. The injection of tuberculin always gives a negative result in the early stages of this latter disease, and this, with the histological examination of an excised gland, is regarded by Osler as quite characteristic. The supervention of tubercular disease of the lung or bones also would settle the diagnosis.

Sarcoma is not so easily excluded, but the discovery of the presence of a primary growth is a valuable diagnostic point, as is also the evidence of secondary growths, which are prone to slowly invade neighbouring structures.

Carcinoma of the lymphatic glands is readily recognised by the discovery of the primary cancerous growth to which the secondary glandular enlargement is due.

Splenic leukæmia is recognised by the marked leucocytosis always present ; whilst in Hodgkin's disease, when this sign is present it only exists to a slight degree, and there are

no myelocytes present, and the splenic enlargement is only moderate. When general enlargement of the lymphatic glands occurs in true leukæmia it is secondary, and only occurs late in the disease.

Lymphatic leukæmia is rarely characterised by great enlargement of the glands, and the leucocytosis is always marked, the increase being mainly in the lymphocytes ; but in the acute form occurring in children the large mononuclears may be abundant.

Splenolymphatic leukæmia, in which disease the spleen and glands are enlarged from the commencement, is closely allied to Hodgkin's disease, and the diagnosis rests upon the characters of the leucocytosis, which shows a relative increase in the large mononuclear white cells, in addition to the lymphocytes and myelocytes.

Syphilitic adenitis is recognised by the presence of the primary indurated sore, the common site of the enlarged glands being in the inguinal region. At a later stage the cervical groups are affected, but never to the extent seen in Hodgkin's disease ; and, moreover, secondary or early tertiary symptoms will be found on examination. Murray points out the value of arsenical treatment in causing a reduction in the size of the growth in Hodgkin's disease, when the diagnosis is doubtful in cases suspected to be of a sarcomatous or malignant disease. The X-rays may be advantageously employed in some cases where the thoracic glands are supposed to be enlarged.

Prognosis.—This is always grave, but a small percentage of cases have been known to end in recovery. As already stated, when the enlargement has become general and the symptoms acute, with the presence of cyanosis or dropsy, the end is not far off. Cases in which the fever is high from the start usually run a very rapid course, and may cause death inside eight or ten weeks.

Treatment.—The early removal of a tumour presenting the features of a simple lymphoma or lymphadenoma affecting one or two of the superficial lymphatic glands may prevent the onset of the disease, since sometimes it seems certain that it originates in this way. To wait in such a case till the diagnosis of Hodgkin's disease has become established is to abandon the patient to his probable fate. Though extirpation of large masses of diseased glands is usually useless, nevertheless it must not be forgotten that recovery has been known to follow such an operation rendered imperative by the pressure which they were exerting upon vital structures.

Drug-treatment is summed up in the word 'arsenic' ; no other drug has proved of any real value. Recovery has followed its administration, under the observation of such unimpeachable authorities as Allbutt and Dreschfeld, in cases

where the disease had advanced acutely to a stage which placed the diagnosis beyond doubt. The drug should be pushed to the farthest limit short of toxic effects. Iodine, bone-marrow, mercury, phosphorus, and other alteratives are of little if any value. The X-rays have recently been tried with promising results.

HYDATID DISEASE

This is the name given to the disorder in man, which is caused by the presence in the viscera of the organism known as the larval or bladder stage of *Tænia echinococcus*—a small tapeworm infesting the alimentary canal of the dog. It is also known as Echinococcus disease.

Etiology and Pathology.—The mature worm, as met with in the dog, measures rarely as much as one-fifth of an inch; it consists of three or four minute proglottides and a head furnished with four suckers and numerous hooks. The terminal proglottis shed with the dog's fæces contains the embryos, which ultimately find their way to the human stomach either through a polluted water-supply or directly from the hands soiled by immediate contact with the dog.

The embryo, having reached the human stomach, becomes freed from its envelope, bores its way by means of its hooks through the gastric or intestinal membrane, and, reaching the blood-stream of the portal system, finally takes up its abode in the liver or other organ. At first solid and surrounded by a thick transparent envelope or capsule, the hooks soon disappear, and the embryo becomes a vesicle or small cyst lined by its parenchymatous endocyst, whilst its outer envelope becomes laminated and continues to increase in thickness as its fluid contents augment. Owing to the specific irritation which the bladder exercises upon the living structures which surround it, a fibrous enveloping capsule is slowly formed from the tissues, which encloses or shuts off the embryo and its own proper capsule and endocyst. It is through this external adventitious envelope that it derives its nutrition.

From the endocyst, which represents the vital part or germinal layer of the parasite, minute hollow buds begin to appear. These, in contradistinction to the primary or mother cyst, are called *brood-capsules*, and they develop within them the heads or *scolices*, each scolex being armed with suckers and hooklets, as in the head of the mature worm, but these are invaginated. At this stage the parasite presents the appearance of one large bladder, or single cyst, which may grow to huge dimensions with thousands of brood-capsules sprouting from its wall, each capsule containing many scolices in various

stages of development, and each usually attached to the interior of its capsule, though by rupture of their stems some may be free.

A more common condition in hydatid disease in man is met with owing to a modification in development by which free cysts form *endogenously* within the primary or mother cyst. These are known as *daughter-cysts*, and spring from infoldings of the germinal or parenchymatous layer of the primary cyst; it is believed they may also arise from the brood-capsules or scolices. In their interior brood-capsules and scolices form, and ultimately they may give rise to other secondary or so-called free grand-daughter cysts, all enveloped by the mother-cyst with its two original layers, and surrounded by the fibrous adventitious capsule.

An *exogenous* variety is also occasionally met with in the human subject. In this form secondary cysts sprout from the germinal layer of the primary cyst, growing outwards. These, upon absorption or rupture of the outer layers of the primary wall, finally develop as independent cysts with separate capsules lying alongside the mother-cyst.

A fourth variety of echinococcus disease is that described by Virchow as the *multilocular* or *alveolar* form; it is seen as one of the varieties of liver hydatid disease, and is sometimes also met with in the bones. In this form the buds, which spring exogenously from the parenchymal layer of the primary cyst, become enveloped by a thick layer of fibrous tissue, which shows on section as a firm stroma containing in its loculi or meshes numbers of secondary cysts undergoing a gelatinous degeneration, the whole being surrounded by a dense thick capsule. In one such case seen by the writer several of these dense masses could be felt in the liver through the abdominal wall, exactly resembling large malignant growths. Verco and Stirling have shown that in some cases the growth may form one single and rounded tumour, whilst in others voluminous cystic groups may develop in the vicinity of disseminated masses of this multilocular form.

These different varieties of hydatid disease in man are all caused by the presence of the same echinococcus parasite, the variations being probably determined by the structural peculiarities in the tissue in which the embryos finally take up their abode.

Sometimes the hydatid bladders are sterile, no scolices or daughter-cysts being present, a condition commonly found in cerebral hydatids. The scolices found in the primary, secondary, or tertiary cysts upon their introduction to the alimentary canal of the dog each finally develops into a mature Tænia echinococcus, whilst a single embryo contained in the terminal mature joint or proglottidis of this worm may give origin to a huge cyst in the human subject, which may

contain millions of scolices each capable of development into a perfect worm after its introduction to the stomach of its primary host.

The fluid found in the cyst presents characters of great diagnostic importance; it is colourless, limpid, free from albumin, and has a s.g. of 1006–1012, and usually contains chloride of sodium and traces of sugar, and sometimes of kreatin and urea. The scolices and hooklets are found present in the liquid in the great majority of cases, and often the fluid becomes bile-stained in hepatic cysts.

After a period, probably to be measured by several years, changes may be observed in the adventitious fibroid envelope or capsule. This may become the seat of calcification by the deposit of lime-salts, which give to it a bony hardness, or it may slough, and profuse suppuration result. The outer covering or ectocyst of the hydatid, which is laminated from an early stage, increases in thickness by the deposition of further layers of transparent, homogeneous, and elastic material.

Various changes are liable to occur in the cyst-contents from spontaneous death of the parasites. The liquid becomes turbid, increases in consistence from a creamy state to the density of a caseated lymphatic gland, and finally may desiccate or dry up and become the seat of a calcareous degeneration like that occasionally noticed in the adventitious capsule, the entire tumour being converted into a firm mortar-like mass, in which remnants of hooklets and fragments of the cyst-walls may still be found. Not rarely is the death of the parasites followed by suppuration and the conversion of the hydatid tumour into a large abscess.

Sometimes the cyst-wall ruptures, and the contents escape into the bile-passages, vena cava, pleura, or peritoneal cavity, with fatal results. This may be the result of injury, or it may follow the formation of pus in the hydatid bladder.

Geographical Distribution of Hydatid Disease.—The affection is comparatively rare in Great Britain, a considerable proportion of the recorded cases being in foreigners who have contracted the disease abroad. It occurs in Northern Germany and Switzerland more frequently than in other European States, in which it is probably as rare as in England, and it is very seldom met with in India, China, or America.

The disease is exceedingly common in Iceland and in Australia. In the former country the intimate relations which exist between man and the dog are accountable for its prevalence, the dog living in the same room and often eating off the same platter as his master. In Australia the sheep, acting as an intermediate host of the bladder-worm, keeps up the supply of parasites, which reach the dog's alimentary

canal either through his worrying the flesh of the animal or feeding upon the offal. The water-supply, often contaminated by the excreta of the dog, is the obvious channel of human infection in Australia.

Age influences the incidence markedly in Australia, the disease being very rare in children, increasing, according to Stirling and Verco, with each decade up to fifty, after which it gradually begins to fall. Men are more frequently affected than women, the opposite being the case in Iceland.

As regards the distribution of hydatids in the human body, the liver, according to all authorities, is affected slightly more frequently than are all the other organs or parts of the body added together, the lungs and kidneys coming next.

Symptoms and Diagnosis.—Symptoms are often latent for a long period, during which the patient maintains perfect health, and in some instances no suspicion of the presence of an hydatid is aroused till rupture or suppuration in the cyst first directs attention to it. The symptoms will obviously vary with the site of the tumour. When this permits of a slow uniform expansion of the bladder-like growth, no symptoms of any kind may be noticed till the weight of the tumour causes a dragging sensation; its presence is then easily detected by palpation. Such is often the case in hepatic, renal, and splenic hydatids. Within the unyielding cavity of the cranium the result is different, as severe pressure-symptoms then show themselves early; but if the subject be a growing child the natural growth and expansion of the skull may permit considerable increase of the cyst before compression-symptoms begin to show themselves, and the flat bones may be thinned out and the sutures widely separated before grave symptoms appear. When the growth increases to such an extent as to finally produce pressure upon vessels, nerves, or neighbouring organs, the symptoms, usually slight, will naturally vary with the structures involved. A marked feature in nearly every case which is recognised early is the slow growth and long duration of the tumour. When suppuration in the cyst-contents or in its adventitious capsule has occurred, the symptoms and signs will be those of an acute abscess. Should such an abscess burst into the peritoneal cavity a rapidly fatal form of perforative peritonitis will be set up. Sometimes in pulmonary hydatids the rupture of the cyst into the bronchial tract demonstrates the nature of the case by causing free expectoration of the cystic contents. Much has been said about the so-called hydatid fremitus or thrill, which has been held by some authorities to be pathognomonic; it is doubtless often present, and is supposed to be caused by the vibration of the free daughter-cysts when the tumour is held in the left hand and percussed by the fingers of the right. An identical sensation has been experienced

during the percussion of other tense elastic cysts filled with limpid liquid. An hydatid cyst to the touch will feel like any other elastic, thin-walled, and tense bag of fluid: it will be smooth, uniform, spherical, or ovoid, and so full that its walls are on the stretch and may not give the usual sign known as fluctuation. With a thickened capsule it may be so hard as to be mistaken for a solid fibroid, and when covered by a thick layer of the tissue in which it has developed a diagnosis is impossible unless by aspiration. This, though dangerous in the lung, may under precautions be safely accomplished by a fine hypodermic needle thrust gently into the cyst when it is situated in the liver. Only a small quantity of fluid should be removed, under strictest aseptic conditions. The presence of hooklets, scolices, or shreds of laminated membrane is absolutely characteristic. Renal cystic or hydronephrotic and cerebro-spinal fluid may be mistaken for hydatid liquid when hooklets and scolices are absent, but the presence of a considerable amount of urea or uric acid in the former, and of a cupric-oxide reducing agent in the latter, assist, if they do not clinch, the diagnosis. Moreover, the s.g. of hydronephrotic fluid is often higher than that of hydatid fluid.

The yellow viscid liquid from an ovarian cyst is albuminous and may have a density as high as 1045, and it contains pseudo-mucin. A distended gall-bladder yields a fluid which is viscid, somewhat but not necessarily bile-stained, and always contains mucus; whilst a cyst of the pancreas may usually be recognised by the presence of cholesterin in company with the pancreatic ferments. The hydatid cyst contains a ptomaine which, though not recognisable by chemical tests, may be demonstrated by the appearance of an urticarial rash after rupture or puncture of the cyst has occurred owing to absorption of some of the extravasated liquid.

Recently Welsh and Barling have investigated the leucocytosis which is often present in hydatid disease. These observers found a marked eosinophilia in about half the cases examined, which rapidly disappeared (within a few hours) after operation. They also found a basophilia in about half the cases, which, however, bore no relation to or correspondence with the eosinophilia—a fact which they consider of diagnostic importance.

Treatment.—The reader will find the various methods fully discussed in the author's 'Dictionary of Treatment' (4th ed.), pp. 421–424. Prophylaxis is secured by scrupulous attention to the purity of the water-supply in districts where the dogs are infected, and to rigid cleanliness of the hands which come into contact with the coat or tongue of the animal.

All anthelmintics are valueless; electrolysis, acupuncture, and injections are dangerous and generally useless. Aspiration of a small quantity of the fluid by a fine needle may cause the

death of the parasite and the shrivelling up of the cyst and its contents, but, owing to the danger of suppuration following, the operation is being abandoned for more radical measures.

These consist in a free incision made over the most prominent part of the cyst, which is then turned out, the margins of the cyst-wound being stitched to the skin-wound, so as to secure drainage ; or the margins of the wound in the cyst-wall are stitched together and dropped back into the abdominal cavity, which is then permanently closed by suture. Suppurating cysts are treated by either of these methods, but the former (known as Lindemann's operation) is preferable, whilst the latter (or Bond's operation) is more suitable for non-suppurating cysts.

HYDATIDS OF THE LIVER

Symptoms.—These may remain latent, their presence often being only discovered in an examination after death from some other disease. The presence of hydatid disease, even when extensive, does not usually interfere with the health or nutrition of the individual till suppuration or rupture of the cyst occurs, or unless, in those cases where vital organs are subjected to the pressure of the growing tumour, as in the brain.

The tumour by its mere bulk or weight may produce a sensation of dragging, rarely amounting to actual pain, in the hypochondrium or right scapular region.

Physical Signs.—There may be no evidence of a tumour on examination if the cyst be moderate in dimensions and seated deeply in the hepatic tissue. Large deeply situated hydatids may produce great and apparently uniform enlargement of the organ, the dulness extending upwards beyond the nipple and downwards as far as the iliac crest ; but usually bulging in some particular direction may be localised on careful percussion and palpation. Bulging may be observable when the upper part of the right lobe is the seat of the growth, the lower ribs being pushed outwards and the intercostal spaces widened, the dulness extending upwards to the second or third rib. In hydatids originating in the left lobe, the heart may be displaced considerably upwards and to the left ; but if the site of the tumour is well towards the spine, the cardiac organ may be pushed forwards and the physical signs of hypertrophy may be closely simulated.

When the tumour is situated upon the anterior surface near to the margin, the physical signs are obvious, the cyst being easily palpable ; it is tense and elastic, though not usually exhibiting fluctuation, and sometimes the hydatid thrill can be elicited by grasping the growth with the left hand and tapping smartly with the finger-tips of the right.

Occasionally some tenderness may be present over the tumour owing to a localised peritonitis, or stretching of the serous covering in rare cases where its growth has been rapid.

The margin of the liver is uniform and smooth, and the organ feels firm and elastic ; where numerous cysts are studded throughout the liver, some may be felt along the margin, forming nodular masses closely simulating malignant growths. In one rare case, already referred to, an enormously enlarged liver was felt by the writer to be studded over with great hard irregular masses, such as are only to be seen in the most advanced cases of hepatic cancer ; these were the dense massive growths produced by the rare multilocular form of hydatid disease, and they were present in a young patient in apparently typical health and robust condition. Not seldom one or more cysts may be felt hanging down from the lower surface of the liver like distended gall-bladders. Ascites and jaundice are so rarely present that they form no part of the clinical picture unless rupture or suppuration has occurred.

Suppuration may follow after injury or tapping ; it often occurs without apparent cause, but not unfrequently it can be traced to the death of the parasites caused by bile finding its way into the mother-cyst. After the formation of pus in the cyst-wall or in its interior, as a rule the symptoms become acute, with high fever, rigors, and sweats, accompanied usually by jaundice and all the physical signs of an hepatic abscess, which, if not speedily evacuated, may burst into the biliary passages, peritoneal cavity, stomach, intestine, pleura, bronchi, vena cava, or elsewhere.

Diagnosis.—This is often a matter of great difficulty, unless the hypodermic needle be inserted and a small quantity of the hydatid liquid withdrawn. Even in those cases where the cyst is sterile and the hooklets are absent, the chemical examination of the fluid will settle the diagnosis, the low s.g., absence of albumin, &c., being in the case of liver hydatids conclusive. The only apparent exception is :

Distension of the gall-bladder.—In this condition, in rare cases, the fluid may be watery and colourless if it be not bile-stained, but there is always some albumin and mucin present, though they may be in small amount. A distended gall-bladder will closely simulate a hydatid cyst which depends from the lower surface of the liver ; but the latter is extremely unlikely to occupy the exact site of the gall-bladder, and it usually is not of the characteristic egg-shaped, lemonade-bottle configuration, and as a rule it is not so freely movable as the distended gall-bladder.

If such a tumour has already suppurated it can only be distinguished from a purulent collection in the gall-bladder by the discovery of hooklets, scolices, or fragments of the laminated cyst-wall in the contents drawn off. A history of

previous attacks of gall-stone colic is of great value in the diagnosis.

Malignant disease of the liver may resemble hydatid disease where several cysts are scattered through the organ near to its lower margin, and especially when the bossy tumours of the rare multilocular form are palpable. The absence of cachexia, ascites, jaundice, pain, tenderness, and wasting, the early age of the patient and the slow clinical progress of the disease, all negative cancer ; but if suppuration has already occurred in one of the cysts, cachexia and emaciation may supervene, and the only distinguishing feature may be the youth of the patient.

Syphilitic disease of the liver may be differentiated with difficulty by the physical signs when the fibroid deformities caused by old gummata can be felt through the abdominal parietes. There is, however, never in hydatid disease the diffuse thickening and gluing of the liver to the abdominal walls and other organs so commonly found in syphilitic livers, and this perihepatitis is usually associated with great hardness, tenderness, and pain over the irregularly enlarged organ. The recognition of other syphilitic lesions and of a history, and the absence of the characteristic fluid on tapping, will settle the diagnosis of syphilitic perihepatitis.

Fatty and lardaceous diseases of the liver are distinguished by the uniform enlargement of the liver, by the history of the case, and by the presence of these diseases in other organs as in the kidney.

Hypertrophic cirrhosis and the chronic congestion produced by *valvular lesions* cause uniform enlargement of the liver, which is accompanied with more or less general tenderness and some degree of jaundice, and always in the heart-cases with much ascites.

Hepatic abscess, when of the so-called tropical form, may present exactly the same physical signs as a suppurating hydatid cyst, and can only be differentiated by the history and by the use of the aspirating needle, which will afford evidence of hooklets in the one case and not in the other. A history of previous dysentery is the rule in abscess.

Cystic disease of the liver, independent of hydatids, is extremely rare, and can only be differentiated by aspiration ; the presence of great cystic enlargement of both kidneys and of the spleen, in conjunction with a cystic liver, would be in favour of the simple disease, though, as remarked by Verco and Stirling, multiple hydatids might be present in all these organs. In the multilocular case already referred to the huge hepatic tumour was diagnosed by a very eminent authority to be a simple cystic disease of both kidneys.

Hydronephrosis usually begins in the loin and gradually extends upwards. If there be a history of calculus, and if the

tumour suddenly disappears, and this disappearance be accompanied by the expulsion of a great amount of urine from the bladder, the diagnosis is obvious. In many cases, when first seen the hydronephrosis already occupies the site of an hepatic hydatid, and the differentiation is extremely difficult because the fluid obtained by the aspirating needle may in some rare cases be non-albuminous, colourless, very limpid, and of low s.g., and may only contain traces of urea. The diagnosis must therefore rest mainly upon the presence or absence of hooklets, scolices, or fragments of laminated membrane. The hydronephrosis moves but slightly during respiration, and the colon can usually be made out in front of it.

Right pleural effusion is not likely to be mistaken for hydatid disease, but a large cyst in the upper part of the liver at the posterior aspect may push up the diaphragm and pleura, and be mistaken for pleuritis with effusion. Little importance need be attached to the curving of the upper limit of dulness; the diagnosis must be made by careful puncture with a fine hypodermic needle, if the history does not establish it, which it seldom does.

Empyema may result from the rupture of the cyst into the pleura, and a careful examination of the pus which forms will give evidence of hooklets, &c.

Treatment.—The only method now considered justifiable is that already described—a free incision and the removal of the cyst with its contents, without attempting to establish previous adhesion between the cyst-capsule and the abdominal parietes.

HYDATIDS OF THE LUNG

These, like liver-hydatids, may give rise to no symptoms or signs for long periods; indeed, as a rule, unless the cystic tumour be of great dimensions, no suspicions of the presence of a cyst are aroused till rupture of its contents takes place and the expectoration reveals shreds of membrane, secondary cysts, &c. Dyspnœa, cough, pain, and hæmoptysis are the principal symptoms met with before the rupture of a large pulmonary hydatid tumour, and these are the result of the pressure of the growth. The physical signs at this stage are in the main those of pleuritic effusion, but local bulging upon close examination may reveal the nature of the case. The signs are practically identical with those produced by a pleural hydatid, which, however, is extremely rare as a primary affection.

The thin adventitious capsule of a pulmonary hydatid is very liable to spontaneous rupture, in which case, if it opens into a large-sized bronchus, symptoms of severe suffocative catarrh may suddenly supervene. After the expulsion of the

contents of the cyst, the physical signs of a large cavity may be recognised. More frequently, however, the cyst does not rupture till inflammatory mischief has been established in the surrounding pulmonary tissue, and the first results of rupture may be the expectoration of purulent fetid material which has been expelled from the cyst, whose contents have become gangrenous. In either case sepsis supervenes, and the cough becomes severe and chronic, quantities of foul pus and shreds of decomposing membrane, mixed with daughter-cysts and hooklets, being brought up from time to time, the case soon presenting the clinical picture of pulmonary gangrene, chronic phthisis, or of bronchiectasis. Such cases are after a time prone to end in fatal exhaustion if not carried off by a profound hæmoptysis.

The symptoms are of the same nature in those cases where the original site of the tumour is in the upper part of the liver, and the cyst after invading the lung ruptures into a bronchus. In not a few cases the pulmonary hydatid bursts into the pleural cavity and produces a purulent pleuritis or localised empyema, as is also frequently the course of an hepatic cyst.

Diagnosis.—*Phthisis* is eliminated from the diagnosis with ease if the case come under observation at the stage when the daughter-cysts and other characteristic contents are being coughed up; and it is necessary to remember that at this period the physical condition of the patient may be one of robust health. The same remark will apply to those cases where some cough and hæmoptysis precede the rupture of the hydatid; the absence of fever and of tubercle bacilli from the sputum, and the site of the dulness produced by the cyst, which is very rarely apical, are also important points in the diagnosis. At a late stage the diagnosis is very difficult, and will depend upon the basilar site of the hydatid cavity and the absence of bacilli in the sputum.

Bronchiectasis and *gangrene* are differentiated, the former by its very chronic, and the latter by its acute progress, and both by the absence of membrane, daughter-cysts, or hooklets in the sputum at any stage of their career. In all cases of doubtful diagnosis the discovery of an hydatid cyst in some other part of the body will clench the diagnosis. The recognition of bile in the sputum is a valuable point in the diagnosis of pulmonary and pleural hydatid disease originating in the convexity of the right lobe of the liver.

Pleural effusion can only be excluded by the use of the hypodermic needle.

Treatment.—If seen and recognised before rupture, the tumour should be cut down upon and portions of the overlying ribs excised, so as to permit of the complete removal of the cyst. This is unquestionably a safer plan than any attempt to cause shrivelling up of the growth by aspiration, which is

much more dangerous here than in the abdomen. When rupture or suppuration has already occurred before the patient is seen, the immediate danger of suffocation from the cyst-contents has passed away, and the physician can form an opinion by the amount of hectic fever, fœtor, and gangrene present whether an immediate drainage of the cavity through an external incision is demanded or not. Undoubtedly some cases spontaneously recover after the coughing-up of all the *debris* of the cyst walls and contents; but many others will succumb unless prompt surgical measures be resorted to before the patient's strength is too far exhausted. Hepatic hydatids which open into the lung have a heavy mortality, but even in most unpromising cases complete success has followed the opening of the thorax and the draining of the hepatic abscess through an abdominal incision made at the same time.

HYDATIDS OF THE KIDNEY

If the cyst has not ruptured, it will cause no inconvenience to health save by its bulk when it is of large dimensions. Moderate tumours have shrivelled up and disappeared, but the most usual course is rupture into the renal pelvis, in which case the contents will appear in the urine, often producing intense renal colic on their passage through the ureters, and painful micturition, and even urethral obstruction. Pyonephrosis may result from suppuration of the cyst, or pus may appear in abundance in the urine.

Diagnosis.—The diagnosis of a large renal hydatid from a *calculous hydronephrosis* is sometimes an impossibility even after aspiration by the needle, owing to the similarity of the fluids; for, as already mentioned, the renal like the hepatic hydatid may be sterile, and in rare cases the hydronephrotic liquid may contain the merest trace of urea, and be deficient entirely in albumin. In such a case light may be forthcoming from the history; the passage of calculi at a former stage, and the periodical discharge of large amounts of urine concurrently with a temporary disappearance of the loin tumour, are almost as conclusive evidence of hydronephrosis as is the passage of hooklets, &c., in hydatid disease.

Renal tuberculosis is often associated with pyuria, and occasionally with pyonephrosis. The differential diagnosis from a suppurating renal hydatid will depend upon the discovery of the presence of the tubercle bacillus in the urine and the absence of hooklets, &c.

Treatment.—This is the same as for hepatic hydatids: the cyst should be cut down upon and completely removed, free drainage being provided for. In cases where rupture has taken place and the mechanical obstruction of the passages by the cyst-contents is not complete, whilst health remains robust,

operative measures should be postponed with the hope of spontaneous recovery, which often occurs.

HYDATIDS OF THE BRAIN

These are comparatively rare even in Australia and Iceland, not more than 4 per cent. of all hydatid growths being found inside the skull. The cyst is generally situated in the cerebral hemisphere, very rarely in the cerebellum.

The symptoms are those described under 'Cerebral Tumour,' and the diagnosis can only be established by the discovery of hydatid disease in some other portion of the body, or by trephining; and this latter, with the establishment of drainage, has been resorted to successfully as a means of radical cure.

Hydatid disease of the spleen, mesentery, and peritoneum is still rarer than the cerebral form, but much more amenable to radical removal. Spinal-cord and heart hydatids are only of pathological interest; the disease, when it occurs in bone and other tissues, belongs to the domain of surgery.

HYDROA. (See DERMATITIS HERPETIFORMIS, p. 267.)

HYDROCEPHALUS

This term is usually employed to designate the conditions in which accumulations of fluid occur within the cranial cavity.

Acute hydrocephalus is a term still often used as a synonym for tubercular meningitis, and sometimes for ordinary acute or basilar meningitis, from whatever cause. It will not be used in either sense in this article, since both of these affections are here excluded from consideration.

Hydrocephalus is sometimes spoken of as *external* when the effusion is confined to the subdural space, and *internal* when the fluid has accumulated in the ventricles of the brain. It is this last form which possesses clinical importance, and hence hydrocephalus is simply defined by some authorities as a dropsy or effusion into the ventricles, ignoring the external form, which is always secondary to cortical wasting or arrested development of the brain, in which the diminished bulk is made up by fluid to fill the vacant space. Before dismissing this external form, which is a common result of senile degenerative changes which tend towards atrophy of the cerebral mass, it may be mentioned that it occasionally occurs before birth and causes great enlargement or expansion of the skull; this rapidly progresses, should the infant be born alive; thus a picture, though a rare one, is presented which is clinically identical with the following common and important type of the disease:

Chronic hydrocephalus, or chronic internal hydrocephalus, is usually a *congenital* disease; there is, however, an *acquired* form which may occur at any age.

The *congenital form* is usually evident at birth, or noticed very soon after; sometimes it causes such obstruction during labour as necessitates perforation of the cranium or death before delivery. Commonly the enlargement of the skull, though present at birth, is not obvious, but rapid increase in the circumference soon becomes very marked and progressive.

Etiology.—The causation of the disease is obscure: injury to the mother or fœtus produced by blows or falls, prolonged grief or worry, chronic alcoholism, and syphilis have been suspected. In a considerable proportion of cases hereditary predisposition has been observed, several children of the same parents being affected, and sometimes a similar history may be obtained in the family records of the parents.

Pathological Anatomy.—The ventricles are distended by fluid, sometimes to the extent of several pints in long-standing cases; all the cavities are involved, but the fourth ventricle least of all, the lateral ones suffering most. Occasionally the dropsy may be confined to a single chamber when the communications are blocked.

The fluid is colourless and transparent, of low s.g.—1002–1009; there are but traces of albumin, cholesterin, and urea, and some chloride of sodium. The brain-substance is thinned out in proportion to the amount of effused fluid, the convolutions are flattened and completely obliterated, the walls of the cerebral hemispheres enclosing the fluid being sometimes not more than one-sixth of an inch in thickness. The central ganglia are flattened sometimes beyond recognition.

The lining membrane of the ventricles usually appears somewhat granular and slightly indurated, and inflammatory thickening of the choroid plexus is sometimes observed. Though the latter changes are not usually well marked, and are sometimes absent, they have afforded an explanation of the pathological state in the theory that the effusion is the result of an inflammation of the ventricular ependyma.

The changes in the cranial bones are very remarkable; they are attenuated to the thinness of parchment, and the circumferential measurement of the head at the end of the first year may have increased from the normal 18 in. to 36 in. The sutures are widely separated, the fontanelles large and bulging, and the shape of the cranium rounded.

Symptoms.—The signs of the disease are obvious from the above-mentioned enlargement of the skull. The frontal and parietal eminences are very prominent, which, with the increase in the circumference, causes the face to look small; the eyes are pressed downwards by the displacement of the orbital plates; often squinting, and sometimes blindness may

be present. The skin over the head is thin, the veins large, and the hair scanty ; the papery feel of the bones when pressed on is very characteristic, and the head may even be seen to show some degree of translucency when a strong light is placed on one side of it.

Intelligence is very often deficient, evidence of idiocy soon manifesting itself. Occasionally, however, the mental state is normal.

Motor disturbances, as shown by weakness in the limbs, are common, and a spastic condition of the legs may supervene. Convulsions are frequently observed, epileptiform attacks alternating with coma being common, and optic atrophy or choked disc from pressure is seen. In the early fatal cases, club-foot, spina bifida, cardiac and other malformations are not rare.

Death often occurs during the first few months of extra-uterine life ; but occasionally the patient survives, and the enlargement of the head becomes arrested, and even old age may be attained in rare instances. The cases in which the cranium has been stated to decrease in size are possibly to be explained by the natural growth of the face, which renders the disproportion between face and skull less obvious. The writer has had one case under observation for several years, during which the circumference of the head has remained stationary and the intelligence normal, the patient being now about eighteen years old.

In some instances spontaneous rupture and evacuation of the dropsical fluid through openings in the bones entering into the formation of the nasal cavity have been followed by recovery, and in still rarer cases spontaneous perforation of the tissues at a fontanelle or open suture has occurred.

Diagnosis.—*Rickets* is differentiated by the square or box-shaped deformity of the skull, which is very different from the rounded or globular enlargement of hydrocephalus, by the absence of bulging of the fontanelles, and by the manifestation of the changes always present in the other bones.

Osseous thickening of the cranial vault sometimes results from congenital syphilis, but can only be confounded with a case of hydrocephalus, in which the enlargement is moderate. The degree of thinness of the cranial bones may be judged by the intensity or distinctness of the fremitus conveyed to the physician's hand placed upon the scalp when the child cries. Often in hydrocephalus a distinct blood-murmur may be heard on auscultating the scalp, and occasionally in great accumulations an obscure sense of fluctuation may be elicited when the bones are thinned out to a papery consistency.

Prognosis.—This is always most grave, and even in slight cases much uncertainty must always exist regarding the degree of mental impairment which may result in those who survive infancy.

Treatment.—Drugs which remove fluid by the kidneys, skin, and bowels have been tried without any benefit. The only advantages obtainable by internal medication are occasionally seen after cod-liver oil and mild courses of iodine and mercurials; these are, however, slight at the best.

Puncture of the dilated ventricles through the scalp with antiseptic precautions, and the removal of small quantities of fluid at a time, have been occasionally successful, especially when elastic pressure has been continuously applied afterwards to the cranium; but the operation has been so often followed by disastrous results that as a routine procedure it cannot be recommended without serious consideration.

Trousseau's plan of compressing the skull by means of a series of strips of lead-plaster, and the more modern method of pressure by a rubber bandage, seldom do good, and may cause fatal compression. Quincke's lumbar-spinal puncture, though not dangerous, is generally futile, and attempts to establish permanent drainage are usually fraught with considerable danger; the ancient plan of applying heroic counter-irritation to the scalp is worthless.

ACQUIRED HYDROCEPHALUS

This is usually *secondary* to some lesion which produces pressure upon the veins of Galen, as in cases of tumour. It is a common result of an attack of simple basic meningitis which causes obstruction of the foramen of Majendie or other openings, and serous accumulation in all the ventricles, or blocking of the foramen of Monroe may distend the lateral chambers. In this form the disease is of the chronic type, and may appear at any age, but in basic meningitis the patients are rarely more than twelve months old.

Acquired *primary* or idiopathic chronic hydrocephalus is believed by some authorities to exist, but probably in all cases there is some primary obstruction of the foramina by tumours, inflammatory products, or parasitic cysts. In the present state of our knowledge it seems best to regard all cases of chronic hydrocephalus of the acquired variety (*i.e.* all those which do not fall under the congenital type) as being secondary to some obstruction of the foramina or iter, or to pressure on the veins of Galen. (See under 'Meningitis, Acute Simple of Childhood.')

The signs are most variable, differing from those seen in the congenital form because of the less yielding state of the cranial bones. Thus there may be an extreme degree of ventricular dilatation without any evidence of cranial enlargement, unless the subject be a child in whom the bones are yielding. Occasionally, however, the sutures may separate in adult patients, as witnessed by Gowers in tumour-cases where the bones had become previously thinned.

Mental failure, blindness from pressure on the chiasma, convulsive seizures, motor weakness, and contractures, with spells of coma, go to make up a clinical picture not very different from that seen in the congenital variety of hydrocephalus. The progress is not uniformly progressive, there being periods in which the symptoms seem to be stationary or in abeyance. The condition is, moreover, believed to be in rare instances susceptible of spontaneous cure, though death is the rule in one or two years, or even earlier. Many of these cases are incapable of diagnosis from cerebral tumour, the headaches, optic neuritis, comatose periods, and motor disturbances being practically identical.

Treatment.—This is as unsatisfactory as in the congenital form, but where a syphilitic taint has caused the primary lesion success may be accomplished by a prolonged course of iodides in very large doses, and mercury may be tried in conjunction with it. Surgical methods of draining the ventricles are referred to under 'Meningitis, Acute Basic.'

Quincke's Acute Idiopathic Internal Hydrocephalus has been described as arising from a serous inflammation of the lining membrane of the ventricles akin to what occurs in pleuritis with serous effusion, the exudation being supplied chiefly from the choroid plexus; hence he calls the affection 'serous meningitis of the ventricles.' The pathogeny of this form of hydrocephalus is regarded by Mayer as probably parasitic—a view opposed to that of Quincke, who holds that the exudation is non-parasitic, being of the nature of the acute circumscribed œdema seen in the rare cutaneous disease—angioneurotic œdema, which also attacks mucous membranes, and which is probably of the same nature as acute urticaria.

Symptoms.—In very acute cases the symptoms closely simulate those seen in basilar or purulent meningitis and in acute tuberculosis of the meninges, or they may closely resemble those produced by cerebro-spinal meningitis. The fever is, however, trivial, or it may be absent; the cephalalgia and retraction of the head are not so severe, but the optic neuritis is a prominent sign. The knee-jerks may be absent, and the coma is not usually pronounced.

Such cases may recover after several weeks, and they afford an explanation of a percentage of examples of basic meningitis which end in recovery, since a differential diagnosis, without culture-experiments in the examination of the cerebro-spinal fluid, is impossible.

In some instances of this serous meningitis the course is towards chronicity, with intermissions, and the symptoms then closely simulate those of tumour, optic neuritis being constantly present, with intense headache, vomiting, paralysis of eye-muscles and exophthalmos, blindness occur-

ring from pressure on the chiasma by the distended third ventricle. The pulse is markedly slow or much quickened; motor weakness and spasms are common. Mayer observed in several cases a general rapid vibrating tremor accompanying active movements, and he emphasises two features of importance in the differential diagnosis—*i.e.* that the disease is often associated with a congenitally abnormal shape and size of the skull; and secondly, that remissions and intermissions of years occur, which is very uncommon in cerebral tumour. Focal symptoms are usually absent, and when they appear they are not progressive as in tumour. As stated upon a previous page, it is probable that these chronic so-called idiopathic cases are not idiopathic in the true sense, but are secondary to some primary obstructive lesion; and from this point of view, therefore, they become identical with the ordinary chronic acquired hydrocephalus described upon page 661.

An external primary or idiopathic hydrocephalus has been described with symptoms similar to the internal form, but it is very rare.

Prognosis.—The outlook is always serious, but complete recovery is not rare, even after the appearance of headache, vomiting, and optic neuritis. In the cases resembling tumour, which run a chronic course, the prognosis is grave, but occasionally recovery has been observed, often, however, with damaged sight or other permanent injury.

Treatment.—Lumbar puncture has been followed by complete and permanent relief in the acute cases; the fluid is colourless and transparent, flows under high pressure, and is albuminous—characters which are in no way diagnostic, Puncture of the ventricles gives immediate relief in most cases and is sometimes curative. Quincke always employs mercurials whether lumbar puncture be resorted to or not, and with these he recommends counter-irritation by means of tartar-emetic ointment.

HYDRONEPHROSIS

This term is employed to designate a condition in which there is atrophy of the renal substance, with dilatation of the pelvis and calyces of the kidney, caused by over-distension with retained urine or weak saline fluid, the result of a mechanical obstruction in the ureter, bladder, or urethra.

It will be seen from the above definition that hydronephrosis may be present with the ureters permeable and even widely dilated, the communication between the kidney and bladder being quite open. Usually no tumour can be felt under such circumstances, except in some congenital cases

where micturition is impossible, when the affection will obviously be bilateral.

It is with the less common condition that the physician has to deal, in which the kidney is distended, owing to the blocking of one ureter, so that a quantity of retained fluid accumulates behind the obstruction and forms a palpable tumour.

Etiology.—A considerable number of these tumour-cases are of *congenital* origin, and are the result of abnormalities in the ureters or of imperforate urethra. They are therefore often bilateral, and may cause death at a very early stage of infant life ; sometimes the tumour has reached such dimensions before birth as to render delivery impossible till tapping of the infant's abdomen has been resorted to. Often other abnormalities are also present.

In the *acquired* form the blocking of the ureter is usually unilateral where a palpable tumour is present. The obstruction is often the result of the impaction of a calculus, ulceration or growths of the bladder involving the orifice of the ureter, cancerous tumours of the female pelvic organs, and twisting of the ureter from movable kidney. Prostatic disease, urethral stricture, and various vesical affections will cause hydronephrosis, with much dilatation of the ureters on both sides, usually without the presence of obvious tumour. The condition is much more common in women than in men, and is sometimes met with in pregnancy and in uterine displacements which interfere with the patency of the ureter.

Pathological Anatomy.—The first results of the distension are seen in the widening of the renal pelvis, preceded by atrophy of the pyramids and followed by dilatation of the calyces. These expand into ovoid pouches, the whole covered over by a thin layer of renal cortex, forming a large lobulated tumour distended by fluid, or in long-standing cases the lobular structure may disappear owing to absorption, rupture, or stretching of the fibrous septa between the dilated calyces, and the tumour then will consist of one large uniform smooth bag or cyst, devoid of renal secreting tissue. Often some hypertrophic enlargement of the sound kidney may be observed.

The *fluid* found in hydronephrosis at an early stage will present the characters of diluted urine. Usually, however, by the time the tumour or cyst has become obvious it has ceased to contain urea in any appreciable amount ; it consists of water holding chloride of sodium and traces of other salts in solution, is usually slightly albuminous and pale yellow or colourless, though occasionally it is dark and viscid. Its s.g. is low—1006–1018—and the reaction is faintly acid or neutral. The dimensions of the cyst vary greatly, from the size of a cocoa-nut to that of a bag capable of containing several gallons and filling the entire abdominal cavity like a large ascites or ovarian tumour.

Symptoms and Signs.—These may be entirely absent until the cyst reaches considerable dimensions, and not unfrequently the discovery of its presence is accidental. Usually there is no pain or tenderness, but occasionally large cysts cause much suffering.

The enlargement begins in the renal region; dulness on percussion and fluctuation on palpation are usually obvious, and small tumours can be felt to be lobulated. Careful percussion will usually demonstrate the presence of the colon in front; the fingers may be inserted between the tumour and the rib-cartilages; by pressing upon the loin with one hand the tumour may be felt to move under the opposite hand applied in front, and in this way also a fair idea of its size may be arrived at. It is only slightly influenced by respiration.

The character of the urine is generally entirely negative, the sound kidney performing its functions faultlessly; but one of the most characteristic features in connection with hydronephrosis is *intermittency* in the size of the tumour synchronous with greatly increased flow of urine. This is caused by the cyst emptying itself through the ureter into the bladder, and in some cases the phenomenon is periodic. When the disappearance of a doubtful tumour is associated with marked transient polyuria, if rupture of an hydatid be eliminated by examination of the urine the diagnosis of hydronephrosis is certain. Sometimes blood, albumin, mucus, or pus may be found in the augmented secretion after the disappearance of the tumour. In rare cases it does not fill up again.

Diagnosis.—*Pyonephrosis* can only be differentiated by the use of the aspirating needle when the obstruction is complete and no pus appears in the urine, and when fever, rigors, &c., are absent.

Hydatids of the kidney are distinguished from hydronephrosis by the differential points mentioned on page 657.

Ovarian tumour, differentiated with ease in the early stages, presents a problem whose solution is a matter of great difficulty at a later period. An ovarian cyst first makes its appearance in the lower abdominal region and gradually extends upwards, whilst the hydronephrotic tumour begins in the loin on one side and gradually extends forwards towards the middle line; it has the colon in front of it, whilst the ovarian growth pushes the intestines behind it into the flanks; it is in its early stages much more movable than the hydronephrosis, and it involves the uterus by dragging it upwards or thrusting it to either side. The nature of the fluid removed from an ovarian cyst is very different usually from hydronephrotic fluid, being viscid, greenish-yellow or brown, always albuminous, and contains pseudo-mucin. The liquid from parovarian cysts may be readily mistaken,

since it is usually colourless and watery in appearance, of low s.g., and may contain little albumin.

Ascites is differentiated in the great majority of cases by its history and by the presence of its obvious cause. When seen at a late stage of its career, a large hydronephrotic cyst, filling the entire abdomen and containing many gallons, is very liable to be mistaken for ascites. Aspiration will be conclusive in such a rare case.

Sarcoma and other renal growths, and *perinephritic abscess*, are eliminated by their history and constitutional effects, especially the latter by its rapidity and the severity of the inflammatory action, which produces high fever and other symptoms of suppuration.

Movable kidney may be mistaken for a small hydronephrosis, and it must not be forgotten that both conditions often exist together, the kinking or twisting of the ureter, caused by the movements of the floating kidney, being sufficient to produce complete obstruction. Indeed, so frequently is more or less hydronephrosis found associated with movable kidney that in obscure cases of this latter condition the diagnosis is made by noting intermittency in the size of the doubtful tumour coincident with polyuria. A considerable percentage of all intermittent hydronephroses are caused by or associated with movable kidney. The uncoiling of the twisted ureter affords an easy explanation of the repeated emptying of the dilated pelvis of the wandering organ; though in other cases, not associated with increased motility, a valvular condition of the obstruction in the ureter or the sudden passage downwards of a calculus is usually held to be the explanation of the discharge.

Prognosis.—A unilateral cyst may remain quiescent for an indefinite period without producing ill-effects of any kind; but if the distension be great, rupture may occur. Occasionally suppuration supervenes, especially in recent cysts. The blocking of the sound ureter may, however, occur from the same causes which previously had occluded its fellow, as in calculous disease, and then death must inevitably ensue. In bilateral hydronephroses the wasting of the renal secreting substance may cause death, even when no palpable tumour has been observed.

Treatment.—Small cysts may be left without interference. If there be reason to suspect a movable condition of the kidney, the use of a suitable abdominal binder and pad may remedy the state of affairs; in obstinate cases the movable organ may have to be fixed in its position by sutures.

Occasionally the cyst has been emptied by gentle friction of the abdominal walls, and careful manipulation applied to the tumour, so that the contents have been squeezed into the bladder.

Great accumulations of fluid must be removed by tapping, and sometimes the tumour does not fill again ; but usually several tappings are necessary as a palliative measure, when more radical procedures are contra-indicated. The puncture on the right side is made half-way between the iliac crest and the last rib, two and a-half inches behind the anterior superior spinous process. On the left side the best spot is found in front of the interval between the last two floating ribs.

Nephrotomy should be performed without waiting if the first tapping has proved a failure ; drainage of the cyst must be provided for through the loin-wound. Should the kidney be found hopelessly disorganised, or should the nephrotomy-wound refuse to heal, the operation of *nephrectomy* should be carried out without further delay ; moreover, this operation in suitable cases has a smaller mortality rate than nephrotomy with drainage.

HYDROPHOBIA

This is the name given to the disease in man which corresponds to rabies in the dog or other animal. It is a specific disease produced by the inoculation of a specific organism or virus which abounds in the spinal cord, cerebrum, peripheral nerves, and salivary glands of rabid animals, by whom it is usually transmitted to man through the medium of their salivary secretion.

Etiology.—Rabies, though most frequently met with in the dog, cat, wolf, and fox, may affect any warm-blooded animal.

Children and men are more frequently affected by hydrophobia than women because of the nature of the clothing of the latter, which affords better protection against the teeth of rabid animals, as the inoculation almost invariably takes place through bites usually received upon the exposed parts of the skin. The disease in the great majority of all cases is communicated by dogs ; and in its distribution, though it is a comparatively rare disease, it is as widely spread as the distribution of this animal itself, and statistics prove that the disease might in most countries be eradicated by the rigid enforcement of a universal muzzling order. In Russia the bites of rabid wolves produce a considerable percentage of the cases of hydrophobia met with in some parts.

The specific virus has not been isolated, and its nature, therefore, has never been demonstrated, though it is proven to exist in the saliva. Many organisms have been from time to time discovered in this secretion and in the nervous system, which were supposed to be the specific agents, but the active microbe still awaits conclusive demonstration. All pathologists agree that rabies and hydrophobia must be caused by

a living virus with power of multiplying itself like the tetanus bacillus, and it appears highly probable that this living virus grows more rapidly in nervous tissue, as will be presently mentioned. The symptoms of rabies and hydrophobia are in all probability the result of the direct action on the nerve-centres of the chemical poison manufactured by the micro-organisms.

Incubation.—When an individual has been inoculated with the virus through the bite of a rabid dog, the wound heals usually with rapidity and no evidence of malaise or illness appears for a considerable period. This stage of incubation varies within the widest limits known in any infectious disease ; it may be as short as fourteen days, and certainly as long as fourteen weeks. The instances in which it has been reported to have lasted for three or five years are probably cases in which a second unsuspected inoculation occurred. The average duration of the incubation-period may be accepted as about from six to eight weeks.

Great importance, from a therapeutic point of view, must be attached to the conditions which modify the length of this incubation-period or time elapsing between the inoculation and the first symptoms of the malady. When the bite has been received in some part of the body where the nerve-supply is scanty, as on the skin of the dorsal region or of the arms and legs, the incubation-period is very long. Where nerve-trunks are injured or where lacerated wounds have been caused by the teeth of the rabid animal in regions whose nerve-supply is rich, as in the finger-tips and face or head, the disease supervenes much more speedily, and is always more severe in its manifestations. The incubation is shorter in children as a rule, and it also varies considerably in proportion to an activity of the virus which depends upon some peculiarity in the species of attacking animal. Thus, after wolf-bites it is very much shorter than after dog-bites ; the bite of the cat occupies a position between that of the dog and wolf, both as regards shortness of incubation and severity of the symptoms. Inoculation-experiments in rabbits corroborate the above facts: the short duration, invariable severity, and certainty of success when the virus is introduced directly into the nervous system by trephining the skull, are very striking, and have led to the belief that the poison finds its way by means of the nerves to the brain or medulla after being implanted in the tissues by the teeth of the rabid animal, though undoubtedly rabies may be induced by contact of the virus with unbroken mucous surfaces.

Fortunately the natural inoculation of the virus by the teeth of animals suffering from rabies fails far more frequently than it succeeds. Accumulating statistics prove the truth of Pasteur's calculation that not more than one person out of

every seven bitten by rabid dogs contracts hydrophobia (*i.e.* between 15 and 16 per cent.), though in the case of wolf-bites of the face the percentage must be as high as 80.

Symptoms.—Usually the first symptom to arouse suspicion is an uneasy tingling sensation or pain in the site of the wound, which may show some redness ; in some cases it has been noticed to open up and discharge. In two fatal cases seen by the writer the patients had forgotten that they had received wounds, and their attention was only directed to the seat of injury by the examination necessary for corroboration of the diagnosis. Restlessness, depression of spirits, anxiety amounting to dread, insomnia, with some degree of fever and thirst, soon show themselves. An exalted degree of sensibility or general hyperæsthesia is present; the pulse and respiration quicken; the restless, excited, and suspicious manner of the patient suggest the advent of delirium, which comes on later in short spells of transient rambling, which usually pass off leaving the mind clear ; in some cases the mental confusion is entirely absent. The reflexes are exaggerated and priapism is often present. Reflex spasms are excited by trivial stimuli ; the most characteristic of these is the severe spasmodic contractions of the muscles of deglutition and of respiration which are brought on by any attempt at swallowing liquid to assuage the severe thirst. The spasm produced by an effort to get down a small draught of water may be so severe as to simulate a violent tetanus, asphyxia and even opisthotonos being produced. The dread of this suffering puts the patient into a condition of abject terror at even the sight of water, and hence the origin of the name of the disease. The sound of a running tap or the splashing of water, even a loud noise, a draught of cool air, or a bright light, is sufficient to precipitate a spasm ; sometimes marked maniacal symptoms accompany these spasms, but they soon pass off, leaving the exhausted patient much prostrated and exhibiting a morose and suspicious aspect of mind, in which he will often desire to be left alone. The stories of hydrophobic patients being observed 'to bark like a dog' are exaggerations of the description of the sounds preceding the advent of the laryngeal spasms, or of the sounds voluntarily produced by attempts to clear the throat of the tough stringy mucus, which causes him great discomfort and which he cannot swallow owing to the throat-spasm.

By the end of the third day the pulse begins to fail, the respirations get shallow and irregular, the spasms become more frequent, and death from asphyxia supervenes, often after a high temperature has been reached. Sometimes the progress is a little slower, and inside about twelve hours before the end, symptoms of a general paralysis take the place of the spasmodic convulsions ; a condition resembling the variety

of rabies often seen in the dog, and known as dumb or paralytic rabies, supervenes, and the patient sinks into a comatose state which is rapidly terminated by a sudden failure of the heart.

In young children the picture is more complex and varied, and may become extremely puzzling owing to the absence of all attempts at self-restraint, the symptoms in the early stage of the disease resembling the ebullitions of wicked temper seen in bad examples of 'spoilt children'; and the writer has witnessed attempts at biting the hands of the attendant.

The symptoms of rabies in the dog should not be forgotten by the physician; their diagnosis is of vital importance as regards settling promptly the treatment of persons bitten by suspected animals. The affected dog becomes dull, shy, and suspicious, and often seeks solitude; restlessness soon becomes prominent; the appetite is depraved, and he swallows hay, clothing, bits of wood or small stones or clay, and even devours his own fur, turning away from his usual food. There is no dread of water, but he snaps at everything within his reach, and apparently at imaginary objects; the saliva flows from his mouth, and, though tough, it often appears frothy from admixture of fine air-bubbles. At this stage his gait is characteristic: he often staggers and appears blind to obstacles in his way, and may dash himself against a wall or against a paling, which he at once attempts to seize with a snap and bounds on, very seldom attempting to bite any bystanders unless they be in his direct path. His bark is peculiar, and very different from the usual sound: a series of short loud growls at first, it soon becomes higher-pitched and more prolonged. He rapidly emaciates; marked weakness of the limbs appears, speedily passing into paralysis, which may first show itself in the jaw-muscles by his widely open mouth, which refuses to close when he obviously attempts to bite.

When no longer able to stand he falls upon his haunches; but soon he sinks prostrate, and usually dies before the end of a week or eight days. The so-called 'dumb rabies' begins with paralytic symptoms, which resemble those seen in the last stage of the common furious or maniacal form; the mouth hangs open so that the animal is unable to bite from the first, and death takes place inside three or four days.

Morbid Anatomy.—Though the *post-mortem* changes have long been the subject of laborious research, there is little definite to be recorded. The microscopic hæmorrhages and small cell-growths observed in the floor of the fourth ventricle, in the medulla and cord, the degenerative changes found in the spinal nerve-cells, and the accumulations of leucocytes in the perivascular sheaths, have all been observed in other conditions.

Babès maintains that the changes which he has observed in the nerve-cells in hardened and stained specimens of the spinal cord of rabid animals are pathognomonic. These changes consist in a destruction of the nerve-cells by small embryonic cells which appear around their nuclei. Similar evidence of the destruction of nerve-cells in the ganglia of the sympathetic and spinal nerves, and in those of the pneumogastric, by the so-called rabid tubercles of Babès have been obtained by other observers. The difficulty in the interpretation of these changes lies in the question of how far they may show evidence of alterations in the tissues secondary to increased or altered function, or how far they may be due to the primary action of the chemical poison on the nerve-centres. The virus, though present abundantly in the nervous system, is not demonstrable in the viscera or blood; it has, however, been found in the milk. The true test in all doubtful cases where an animal is suspected of being rabid consists in injecting under the dura mater of a healthy rabbit a small quantity of an emulsion prepared from the suspected spinal cord. When death from rabies follows in less than twenty days the diagnosis becomes certain.

Diagnosis.—This can only be a real difficulty in the early stage of the malady; the presence of the respiratory spasm showing itself on an attempt to swallow liquid, coupled with the history of the inoculation, is quite characteristic.

Tetanus is distinguished by the early appearance of trismus, and the short period of incubation following an injury. In hydrophobia, where the respiratory spasm is sometimes followed by a general tetanic spasm and opisthotonos, there is no trismus, but the so-called *tetanus hydrophobicus* which arises from head-wounds exhibits a paralysis of the facial muscles with stiffness of the jaw-muscles and some difficulty in swallowing. In these, as in all cases of tetanus, the very important element of terror at attempting to swallow, and the other mental symptoms, are wholly wanting.

Lyssophobia, or *pseudo-hydrophobia*, is the only condition liable to be mistaken for the genuine disease. This is but a manifestation of the mimicry of hysteria. It occurs in hysterical persons who have been bitten by healthy animals supposed to be rabid, and in whom the dread of hydrophobia so preys upon the mind that hysterical spasm on swallowing and dread of water, with great mental depression, follow. When the symptoms supervene within a week after receiving the bite the diagnosis is certain; but where they do not appear for a couple of months, and especially if the attacking animal has been known to be rabid, there may be grave anxiety on the physician's mind for a brief period regarding the diagnosis, especially if general hysterical convulsions are present. The absence of fever is of great importance, and the throat-

symptoms on carefully watching the case are seen to be totally different from the true respiratory spasm. The convulsive seizures are often seen to precede the throat-symptoms, and, like most hysterical manifestations, the psychic element of suggestion overdraws the picture: such patients begin to bark and bite, and behave in a manner which is characteristic of hysteria. Moreover, such cases have complete intermissions and are never strictly progressive, and the affection may be suddenly cut short by a calm assurance of the absence of all danger. When the symptoms have lasted beyond a week the possibility of hydrophobia disappears.

Landry's paralysis might be mistaken for the dumb form of hydrophobia, but the history and progress of the case settle the diagnosis speedily.

Prognosis.—Once the symptoms have become evident, an inevitably fatal issue does not admit of doubt. Reported cases of recovery are universally regarded as errors in diagnosis; but one authentic case of recovery where the symptoms of hydrophobia occurred during the Pasteur treatment is recorded.

Treatment.—Preventive treatment is obviously of enormous importance in a disease which is invariably fatal. As already stated, a universal muzzling order strictly enforced would probably eradicate the disease in a short time in Great Britain.

Bites should be treated without a moment's delay by cleansing and the free application of a powerful caustic, and when possible the excision of the lacerated tissue and the conversion of punctured into incised wounds after these have been sucked, when possible by the lips. A tourniquet should be immediately applied when the bite has been inflicted upon a limb.

Pasteurism.—The most brilliant discovery in modern therapeutics was made by the great French chemist who sought and found a way by which an animal or man could be effectively protected against inoculation with the virus of rabies. Recognising the long period of incubation in hydrophobia, his next step was to forestall the symptoms of the disease by a rapid method of immunisation; this he successfully accomplished by inoculating the bitten person with injections of the spinal cord of rabbits in whom rabies had been produced by the virus from dogs.

The details of Pasteur's treatment will be found in the author's 'Dictionary of Treatment,' pp. 429 to 435. Its fundamental principle consists in the immunisation of the patient by means of injections of an *attenuated* virus containing both the chemical poison and gradually increasing doses of the living germs which manufacture it. The first step in the treatment is the providing of a powerful and uniform

deadly virus for the rabbit. This is obtained by passing the poison from a rabid dog successively through one hundred rabbits, when the so-called 'fixed virus' is obtained, which kills a rabbit constantly on the tenth day after subdural inoculation. The spinal cords of rabbits so destroyed by subdural inoculation are dried for varying periods, and these supply the material for the injections into the tissues of persons bitten by rabid dogs. The treatment is commenced soon after the bite has been received, and is concluded in fifteen days; a longer course with larger dosage, known as the *intensive* method, is employed for face-bites and wolf-wounds. The success of the treatment is established beyond all possibility of doubt, and the mortality in persons bitten by rabid dogs and in the more fatal inoculations received by wolf-bites has been reduced to about 1 in 400, instead of a death-rate of about 1 in 4 or 5, or less, which is to be expected when wolf-bites are included. Hence many thousands of lives have been saved by the treatment in the Paris Institute and in similar institutions now established in other countries. The mortality rises with the length of the delay which is permitted between the reception of the bite and the commencement of the injections. When symptoms of hydrophobia arise during the treatment, these cannot be attributed to the injections, since the incubation of the disease has never been known to be shorter than fourteen days, even in face-bites, and this short period of incubation has been recorded only in a few cases.

Serum-therapy on the lines of that so successfully employed in diphtheria is being steadily worked out, and promises to supply a method of treatment much more convenient than Pasteurism.

When the symptoms of hydrophobia show themselves in a patient who has neglected the benefits afforded by Pasteurism, the most that can be hoped from treatment is an amelioration of his terrible suffering by hypodermic injections of morphia, inhalations of chloroform, rectal injections of chloral, or the administration of curare in large doses, together with the most absolute rest and quiet in a darkened room, with one, or at the most two, suitable attendants.

HYPERIDROSIS

This is the term applied to the condition in which there is an excess of perspiration, local or general, depending upon disturbance in the function of the sweat-glands. The term is not usually applied to the sweating which is part of the process of fever and conditions associated with high temperatures, but is commonly reserved for those cases in which the sweating is confined to a limb or portion of a limb, to

regions like the arm-pit, scalp, soles of the feet, and palms, to one-half of the body or to the region supplied by a particular nerve. As regards the half of the body affected, it may be hemiplegic or paraplegic in its distribution.

Etiology.—A general sweating of the entire surface of the body has been observed in hysteria, neurasthenia, and obesity, and would come under the term 'hyperidrosis'; a sweating of the paralysed side in hemiplegia is by no means rare, and it often accompanies the distribution of nerves which are the seat of neuritis. The secretion of the skin is controlled by sweat-centres situated in the cortex, from which fibres pass downwards, decussating at the medulla on their way through the antero-lateral tract to join the spinal sweat-centres. From these, fibres pass out at different levels of the cord, which, passing through the sympathetic, join the cutaneous nerves.

It thus becomes obvious how nerve-disturbance is so frequently associated with excessive sweating, and few cases of affections like ataxia, exophthalmic goître, and migraine but will show at times interesting illustrations of hyperidrosis in some region, if carefully looked for. Even a 'crossed' example of local sweating has been observed occasionally, as where one leg and the arm on the opposite side of the body have been the seat of perspiration.

Symptoms.—The affected region in hyperidrosis feels generally cool, and the amount of sweat varies from that seen in the scant clammy moisture which wets the palms and soles, to the dripping flow which comes from the axilla and saturates the patient's dress. Often the epidermis becomes macerated and its upper layer rubs off, whilst if opposing surfaces of skin are permitted to remain in contact a profuse purulent or serous discharge may originate from the resulting inflammatory action.

The cases of hyperidrosis which usually present themselves for relief are those in which the increased secretion of the skin of the feet and axillæ has an offensive odour, an affection then called Bromidrosis, and described on page 132.

Anidrosis or the opposite condition, where the secretion is absent, may have the same distribution, being also usually due to altered innervation, though it is a general affection in the congenital form accompanying ichthyosis.

Treatment.—Atropine and belladonna will check the excessive secretion by paralysing the ends of the nerves distributed to the sweat-glands, but rarely will such active treatment be required. In the excessive sweating seen in phthisis, these remedies may be occasionally demanded. Crocker recommends as the best all-round treatment the administration of a teaspoonful of milk of sulphur morning and evening.

The rational method of dealing with hyperidrosis will obviously consist in remedying the altered innervation which

causes it, and this can sometimes be accomplished satisfactorily, as in obesity, by attention to dietary, exercise, &c.

Local applications are useful where the increased secretion proves inconvenient, and the astringent agents recommended under 'Bromidrosis' may be selected, in conjunction with perfect cleanliness, frequent bathing of the parts and the use of electricity. Recently the X-rays have been found successful in the treatment of many cases of local sweating.

HYPERPYREXIA

This is the term employed to designate the high temperature reached in many diseases; though there is no fixed or definite demarcation between pyrexia and hyperpyrexia, usually the latter term is applied to temperatures above 106° F. (41·11° C.) The high degree of fever-heat is secondary to the diseased processes in many inflammatory affections, but in some of these the tendency towards hyperpyrexia is much more marked than in others, and this is notably the case in *acute rheumatism*, where a temperature of 108° to 112° has been often recorded. Malarial and yellow fevers, scarlatina, sunstroke, typhus and typhoid fevers, the late stages of tetanus, and hydrophobia, afford frequent examples of hyperpyretic temperatures. Much less frequently is hyperpyrexia met with in pneumonia, tubercle, small-pox, and serous inflammations. In a different category or group should be placed those examples of hyperpyrexia which arise from injury to certain regions of the brain or central nervous system. Thus, section of the spinal cord and injury to the bulb are followed by very high temperature, so also is puncture of the medial side of the corpus striatum, where the heat or thermogenetic centres have been located. The brain-pyrexia observed in these cases and in apoplexy is believed to be different from the true fever-state seen in specific fevers, since rigors do not occur and there is no dilatation of the skin-vessels before the high temperature begins to drop, the fault being chiefly a breakdown in the heat-regulating mechanism. Hyperpyrexia is always a symptom of grave omen, and it is well to remember that as a rule it is entirely beyond the action of antipyretic drugs, being only influenced effectually by cold-water treatment. Its prognosis and treatment will be dealt with under the different affections in which it is liable to arise.

HYPOCHONDRIASIS

This is defined by Gowers as 'a morbid state of the nervous system in which there is mental depression due to erroneous ideas of such bodily ailments as might conceivably be present.'

By most writers it is regarded as a variety of melancholia. By the above definition the condition is differentiated from actual insanity with somatic delusions. In melancholia the predominant characteristic is the morbid *feeling*, whilst in hypochondriasis morbid *sensation* is the main feature.

The borderland between actual insanity and the condition under consideration is very narrow, just as it is between insanity and hysteria; many insane patients are hypochondriacal, and hypochondriacs may present symptoms of melancholia which, of course, bring them into the category of insanity. As Savage states it, the hysterical woman may remain hysterical all her life and never become insane; so the hypochondriacal patient may remain hypochondriacal to the end.

The more severe cases pass into insanity and become hopeless melancholics.

Etiology.—The disease is more common in men than in women, and appears most frequently between the ages of twenty-five and forty-five; occasionally it may be met with in boys.

Heredity is a powerful factor, and in the severe form of insane hypochondriasis a family history of insanity is common. A simple neurotic inheritance is marked in many cases, stamping the patient with a characteristic sensitive temperament which is very prone to develop the habit of introspection, liable to end in simple or insane hypochondriasis. The condition in its minor form frequently originates in some transient disturbed function or diseased action, as in dyspepsia or constipation, and it may exist after these have entirely disappeared.

Symptoms.—The hypochondriac exhibits a morbid anxiety about his general health; he attaches undue importance to the most trivial derangements which generally pass unnoticed in ordinary healthy persons, and finally he usually arrives at the stage in which he is satisfied that he is permanently labouring under one or more diseases.

The mental condition, as it comes under the notice of the physician and not under that of the alienist, shows itself in the first instance in the extraordinary attention which the patient continually pays to his sensations. By continuous concentration of his attention upon ordinary sensations he magnifies their importance and exaggerates their intensity, so that, for example, he becomes constantly conscious of his heart-

beats and of the intestinal sounds produced by flatus, &c. His tongue, pulse, and the various excretions are examined with deep interest or anxiety.

The abdominal sensations, though arising, perhaps, in the first instance, from some trivial and transient error in digestion, remain, and the morbid concentration of attention upon them ends in a distinct *abdominal* type of hypochondriasis.

Another type—the *cephalic*—is that in which the patient complains of abnormal sensations in the head, as a sense of oppression, weight, lightness, tingling, fogginess, &c., sometimes of actual pain or a feeling as if the brain had been constantly strained in making a great mental effort.

Sexual hypochondriasis is a very common form of the disease. Sometimes originating in nocturnal emissions, at all events generally exaggerated by them, the mental condition gradually becomes one of constant depression, and the patient believes that he is impotent; these cases often end in melancholia. Closely allied to this type is the one often met with in which the patient is satisfied that he has contracted syphilis after some occasion of illicit intercourse, and he interprets the normal sensations about the genital organs as evidence of this. Such cases may end in a syphilophobia on the other side of the landmark which bounds sanity.

Diagnosis.—As already stated, the differentiation from *insanity* is a difficult matter in some cases. Gowers lays stress upon the value of the reasonableness of the patient's false ideas. Thus a patient with dyspepsia and acid eructations may convince himself that serious organic disease of the stomach is present, in spite of assurances to the contrary, and in such a case there may be no justification of a suspicion of insanity. The condition is entirely different when the patient becomes possessed of an idea that he has no stomach at all, or that his food passes directly into his head or into some other region of the body. The elimination of real disease is a serious problem in hypochondriasis, and can only be effected after a most exhaustive physical examination, which will afford evidence that the abnormal sensations are independent of any organic basis. The wasting which accompanies abdominal hypochondriasis is very liable to be mistaken for latent *malignant disease*, but the absence of tumour or intestinal obstruction and the presence of free hydrochloric acid in the stomach-contents, with the duration of the symptoms, settle the diagnosis.

Hypochondriasis must not be confounded with *hysteria*, in which the main feature is a psychosis manifesting itself in emotional disturbances and diminished will-power, and associated often with physical signs simulating organic disease, whilst the essential feature in hypochondriasis is the morbid sensations arising in the mind of the patient.

Prognosis.—This is always bad in those whose family history shows a strain of insanity, though it may take a long time before true melancholia develops. In all cases the prognosis is bad when the condition has been of long duration, especially when the patient has already passed middle-life. Upon the other hand, the prognosis is hopeful in young subjects where the symptoms have manifestly originated in some affection which is curable. The tendency of every severe case to end in insanity must never be lost sight of.

Treatment.—The first indication is always to remedy any obvious departure from health which may be found present, as anæmia, constipation, &c. Once the diagnosis is made clear, the best, and indeed the only, course open to the physician is to take the patient into his confidence and explain to him exactly and fully the nature of his ailment. In patients who have already crossed the borderland, and are actually insane, there is not usually anything to be expected from an appeal to reason. To belittle and to treat his symptoms as purely imaginary is but to confirm the invalid in his folly and drive him to further introspection. A considerable percentage of hypochondriacs recover speedily as soon as they have become able to grasp the fact that the distressing sensations or symptoms are due to a perverted nerve-function and do not indicate any organic affection.

The treatment is therefore almost entirely moral, and the patient must be assisted in his own attempts at ignoring, disregarding, or minimising the importance of his sensations by bringing philosophy to his aid. Changes in his environment, as travelling, and the choice of a new and healthy occupation which will engage both his mind and his body at the same time, are serviceable; but above all is the importance of the society of a few discreet friends by whom the exact nature of his ailment is fully understood.

Seldom does the act of prescribing for his sensations accomplish anything but evil to the patient. The physician, when he has once secured the entire confidence of the hypochondriac by a thorough and conscientious examination and investigation of his symptoms, can then often with marked advantage firmly decline to treat him by drugs.

HYSTERIA

It is impossible in a sentence to give any definition which will include the many-sided characteristics of this psychosis, with its motley emotional disturbances and its mimicry of organic disease.

Etiology.—The supposed connection of this affection with uterine disease was the origin of the word 'hysteria,' but the

view is now entirely abandoned. Women are more frequently affected than men, the ratio being variously stated as from twenty to one to a rate of two to one. These remarkable discrepancies in statement are only to be reconciled by a study of the disease in the countries from which the statistics are compiled. Certainly in Great Britain the number of adult males afflicted with genuine hysterical symptoms is very trifling compared with that of women.

Age is a strong factor, most cases beginning between puberty and twenty-five ; about half of the grand total occur between ten and twenty. Before puberty the disease is certainly infrequent, and probably the majority of the early cases occur in male children—a fact which exaggerates apparently the ratio between male and female incidence.

Race is a strong etiological factor, as is evidenced by the great frequency of the disease amongst the French, and especially also amongst the individuals of the Hebrew race, in which male hysteria is very common. Civilisation has a marked influence, since the disease is very rarely observed in savage or partially civilised communities.

Heredity was maintained by Charcot and his pupils as always the prime cause of the disease, and undoubtedly in a very large percentage of hysterical subjects a direct hereditary history is obtainable, some psychic trauma calling into activity the latent hereditary instability.

Congenital predisposition caused by neurotic taint in the parents, in whom no direct history of hysteria can be obtained, is an unquestionable etiological factor in many cases in the offspring. Insanity and habitual drunkenness in the parents are credited also as causes in a small percentage of cases.

Faulty home-training, in which self-indulgence is permitted to retard the development of self-control and of the inhibitory powers, gradually tends towards the formation of a character which is very prone at a later period to manifest itself in hysteria. The evils of injudicious training can hardly be exaggerated in a condition which is mainly a psychosis manifesting itself in a defect of character and emotional instability. The modern practice of the holding of juvenile social gatherings, exposing pampered children to emotional excitement which tends towards the precocious forcing of character, is in many instances deleterious.

Of the exciting causes none are so frequently operative as some profound emotional disturbance—fear, hate, or love playing a part in many instances prior to the outbreak of the first hysterical attack.

Though the necessary association of uterine disease with hysteria is held to be no longer tenable, nevertheless abnormal conditions of the generative organs in both sexes play a considerable part in many instances, and in a small percentage

of cases the affection has disappeared after ovarian or uterine irritation has been remedied.

Sexual excesses in both sexes may be the origin of the disease, and masturbation is undoubtedly a not very infrequent cause; but a more pernicious exciting cause is often observed amongst married couples, where, with the view of preventing pregnancy, the sexual act is voluntarily and purposely never completed. Any serious deterioration of health may precipitate an attack in individuals already predisposed by hereditary or congenital influences or by injudicious moral training. This is not unfrequently seen in the breakdown which follows mental strain in school or college life, though mental strain alone is rarely the cause of hysteria, emotional disturbances being nearly always a factor in such cases owing to the competitive element which is always present.

A factor which cannot be overlooked is the sympathetic imitation or mimicry which exhibits itself in the rapid spread of acute attacks of hysteria in small communities or schools, especially in the presence of some profound religious or other general excitement.

Symptomatology.—This may be considered from the point of view of the symptoms which are present more or less continuously during the intervals between the paroxysms. The paroxysms will be described afterwards, but these continuous or inter-paroxysmal symptoms may in many cases be the only evidence of the disease.

Mental Symptoms.—The mental state or psychic condition is of great importance. It always exhibits characteristic departures, which, though really abnormal, essentially differ from the conditions met with in insanity. The mental power is not affected, and memory is normal, but the *character* is obviously profoundly altered, as is seen in the emotional disturbances in the defect of the will-power over certain movements, and in the inability to resist impulses towards anger, laughter, crying, &c.

Abnormal irritability of temper and variability in disposition are usually prominent features. The emotions are excited by stimuli which in health fall short of producing any manifestations, and when so aroused in the hysterical patient there is a pathological increase in the intensity of the manifestations of the excitement, and a marked tendency in the aroused emotion to rapidly change or alternate with the opposite condition. Thus a fit of violent spasmodic laughter often ends in an explosive turn of crying, and the rapidity of the variations for which no apparent reason is forthcoming is very characteristic, and is not to be explained by the mere loss of inhibition or a deficiency in the power of repressing the emotions, which is also usually a prominent feature.

Self-consciousness is ever present, and the mind of the hysterical patient is always turned inwards upon herself. In this particular there is a constancy which is in marked contrast to the ever-varying disposition of the patient, and the instability of character manifested in the rapid changes of the symptoms of the disease.

The self-consciousness betrays itself in the greed for sympathy, in order to obtain which the patient's symptoms are often consciously or unconsciously exaggerated, and if the sympathy be forthcoming the disease feeds upon it, till the patient's sense of right and wrong becomes so blunted that new symptoms may be feigned. Thus the morbid craving for sympathy may induce the patient to scratch her gums in order to imitate hæmoptysis, or to put chalk upon her tongue to simulate a dyspeptic condition. These perversions of the moral sense are, however, comparatively rare, and are not to be confounded with what one might designate as the real mimicry of the disease. In this latter condition the patient may have no desire to simulate an illness, but, being convinced of a loss of power or of actual paralysis, she becomes unable to exercise the will-power necessary to set the muscles in action, and so may remain bed-ridden with paraplegia for an indefinite period. Being herself thoroughly convinced of the genuineness of her paralysis, she may, however, ultimately be tempted to feign other symptoms in order to convince her friends and her attendant of the reality of the loss of power. Thus the psychic condition may become a puzzle; and in its elucidation it must be always remembered that hysteria is a disease in which often a true clinical picture of an affection is produced of which the patient has never heard, and which could not, therefore, be produced by malingering. This fact is very obvious in those cases in which contractures of a limb persist during sleep. This consideration will assist in the interpretation of those cases in which a diseased condition is exactly simulated by an hysterical patient who has been in contact with a patient suffering from some organic affection, in which case there may be no intention or voluntary desire to feign illness.

Another feature in the mental or psychic condition is the occasional lapse of memory or retrograde amnesia met with after severe attacks. As already stated, the memory of hysterical patients, like that of their general mental power, is usually unaffected, but sometimes after a severe emotional disturbance or 'fit' of hysteria there is left a complete gap in the memory which may wipe out the recollection of a considerable portion of the period of the patient's illness.

The phenomena of delirium, lethargy, and catalepsy, and of the hypnotic and somnambulistic states, with their double

consciousness or dual personalities, will be referred to in the description of the attacks.

Insanity may be associated with hysteria, or may develop after repeated attacks ; it must be regarded, however, not as part of the hysteria, but as a coincidence arising from the common hereditary or neurotic origin. The hallucinatory delirium which accompanies the severe fits is always transitory. The association in the popular mind of hysteria with weak- or feeble-mindedness and insanity is an unfortunate one ; the condition is, upon the whole, more frequently met with amongst the intelligent and educated.

Sensory Symptoms.—These consist of *hyperæsthesia*, *anæsthesia*, and *spontaneous pain* felt in various regions, and affecting the special senses.

1. The *hyperæsthesia* is usually confined to certain circumscribed areas, which may be exquisitely tender on pressure. Sometimes the tenderness is quite superficial, or it may be subcutaneous, and in some cases it is deeply seated, requiring firm pressure for its demonstration.

Hystero-genetic zones are found amongst these tender areas, when firm pressure applied to a sensitive spot produces an hysterical paroxysm or controls a fit already in full force, but every hyperæsthetic area is not a hystero-genetic zone. Spontaneous pain may arise in any of these areas, as well as in the zones. The best marked and most frequently found hystero-genetic zone is the so-called 'ovarian' one—*i.e.* a spot above the inguinal region. It probably has nothing to do with the ovary, since it has been also demonstrated in hysterical males, in whom firm pressure on this region will bring on a paroxysm or induce the sensation of a globus.

The hypersensitive areas are scattered over the body ; the most common one, in the writer's experience, corresponds to the prominence over the spine of the last cervical vertebra. Other sites frequently very tender to pressure are about the middle of the epigastrium, just under the nipple, at the apex-beat, and at the centre of the sternum, more rarely about the vertex and on the limbs. These hyperæsthetic patches are more frequently found on the left side of the body, and in some cases the hystero-genetic zone in the ovarian region, which is here identical with a hyperæsthetic spot, may be present on the left side only. The hyperæsthetic areas are sometimes the seat of a distinct aura, which may usher in a severe hysterical fit or paroxysm.

2. *Pain* of a spontaneous nature—*i.e.* not that evoked by pressure—is an almost constant feature in hysteria. It is very generally, but not invariably, associated with hyperæsthesia, and often its site corresponds to that of some hystero-genetic zone. The head is frequently affected, and the headache may assume the type of the hemicrania met with in

migraine ; at other times it takes the form of clavus, in which an excruciating boring pain is felt in some circumscribed area about the left temple, as if a nail was being driven into the skull ; occasionally the headache is located in the occipital region, in all of which varieties there is usually marked hyperæsthesia.

The cervical region of the spine is often the site of fixed pain, and the dorsal, lumbar, and sacral regions suffer also, sometimes the coccygeal nerves being the focus of a severe coccygodynia. A not unusual region is the hystero-genetic zone found near to the apex-beat, and this may extend to the mammary gland, constituting mastodynia.

Intercostal and facial neuralgic pains are common, but true neuralgia is rare. The pains of hysteria are classed as psychalgias ; they do not correspond accurately to any known nerve-path or district supplied by a given nerve-trunk, but occur in isolated segments or islets. The pains which occur in joints and in limbs the seat of contractures are of the same character. Mayer regards the pains met with in hysteria as of central origin, and classes them as hallucinations of pain caused by direct excitation of the pain-perceptive centres, though they may be also due to slight peripheral stimulation.

3. *Anæsthesia* is even more characteristic of hysteria than the other sensory symptoms, and is usually to be found if carefully looked for, though the patient may be entirely ignorant of its existence. Like pain, it is never found in its distribution to correspond with the ramifications of a particular nerve-supply. Often it takes the form of a well-marked hemianæsthesia involving the head and face, trunk, leg, and arm on one side of the body, stopping exactly in the middle line of the head, neck, and trunk. Throughout the region of anæsthesia sometimes islets of normal sensation may be discovered, and even isolated areas of hyperæsthesia may be found. When appearing on the limbs it usually assumes the distribution of bands or segments. Thus in the upper extremity it may involve the limb from the elbow to the wrist or from the elbow to the shoulder, or the sensation in these parts may be normal while the anæsthesia may affect the fingers and hand, extending some distance up the forearm, assuming the so-called ' glove ' or ' gauntlet ' method of distribution seen in peripheral neuritis. A similar distribution is seen in the lower extremity. It differs from the hyperæsthesia in being rarely distributed in islets or circumscribed areas, and in not being commonly bilateral, and in being found in rare cases to involve the entire body. Like the tenderness elicited in hyperæsthesia, the loss of sensation may be limited to the skin, to the subcutaneous tissue and deep muscles down to the periosteum, bones, and joints. The muscular sense may be involved to the extent of depriving

the patient of a knowledge of the position of his limbs when the eyes are shut.

The sensation of touch, pain, and temperature may be abolished or lessened, or one of these may be affected less than the others. Thermanæsthesia by itself is rare, but analgesia with perfect tactile sensation is common. With the anæsthesia of the body there may be associated—

4. *Anæsthesia of the special senses*, and the association is diagnostic. In a case of hemianæsthesia in which pins and needles may be inserted down to the periosteum without evoking the slightest sensation of pain, the sense of sight, smell, taste, and hearing are all affected upon the same side, which is generally the left half of the body.

The visual disturbance shows itself by the perimeter in a concentric narrowing of the field of vision; the colour-fields are also narrowed, and the power of vision is very easily tired, whilst in rare cases complete blindness in one or both eyes may occur. Generally there is also some contraction of the field of vision on the good side. The senses of smell and taste are blunted, but rarely are they completely abolished, some appreciation of certain odours and tastes being retained. Hearing is affected in a similar manner, the appreciation of certain sounds being retained; but sometimes the deafness is so complete that deaf-mutism is observed. The loss in osseous conduction is often well marked when the deafness is slight, and there may be anæsthesia of the meatus and auricle when the sensation of the skin of the body generally is unaffected. All these deficiencies in the organs of special sense may be marked without cutaneous anæsthesia, and sometimes they may be observable only on the sound side in hemianæsthesia.

Hyperæsthesia of the special-sense organs is sometimes observed; thus the visual field has been in rare cases found to be extended, and the senses of smell, hearing, and taste have been observed to become abnormally acute. The above abnormal conditions of the special senses, like those of the skin and deeper tissues, are characterised by their transient and varying degrees of intensity; they often appear suddenly, sometimes after a severe paroxysm, and they may disappear completely, vanishing without any apparent improvement in the other symptoms, by which points they are easily differentiated from organic changes.

Motor Symptoms.—Various forms of paralysis, spasms, and contractures are common. The paralysis is usually sudden in its onset, following some violent emotion or hysterical fit. It may be hemiplegic, paraplegic, or monoplegic, but the paralysis, which may be complete or incomplete, is never confined to the muscles supplied by any one nerve, and it is not followed by wasting, save such as may result from disuse.

Aphonia, the result of paralysis of the adductors, is the most common form of loss of muscular power met with in hysteria ; it is invariably bilateral, and sometimes in rare cases the tongue participates, so that there is mutism or complete dumbness. Usually, however, the patient is able to speak in a whisper—*i.e.* there is simply loss of voice, which has come on suddenly after some emotional disturbance, and which may disappear as rapidly. Sometimes its onset is gradual, the condition following a laryngeal catarrh in an hysterical subject ; and though the patient is unable to utter a word save in a whisper, she may be able to sing and cough noisily, or scream under excitement. Stammering or stuttering may be observed in some cases, the patient repeatedly pronouncing part of a word in rapid succession, and after a variable interval of silence the remaining syllables may be ejaculated with considerable muscular difficulty or exaggeration.

In monoplegia a limb is more or less completely paralysed, the other limb on the same side being quite free—a state of matters only found in hysteria, and in cases where the nerve of a limb has been subjected to local pressure.

Paraplegia is more common than hemiplegia. Its onset is usually sudden, but incomplete at first ; it gradually deepens till the patient is unable to walk or even to stand. The condition becomes that described as '*abasia*,' in which, with total inability to stand, the movements of the legs are unaffected when the patient is lying on her back or side. More frequently, however, though there be marked resistance to passive movements made in the recumbent posture, the muscular action is not normal, but irregular, or only sustained in an intermittent fashion, and liable to much variation on distraction of the patient's attention.

The loss of power is evidently not muscular, but is owing to lack of energy or will on the part of the patient to bring the muscles into action owing to the conviction that paralysis exists. The paralysed muscles may be freely moved under great excitement, or when the patient is partially under the influence of alcohol or an anæsthetic.

The electric irritability is normal, and, though retention of urine is common, incontinence of urine and fæces never occurs, as in organic spinal affections. The reflexes are generally normal, but often there is some exaggeration of the tendon-reflex associated with a sudden stab of pain in the lumbar region following the tap on the knee-cap. The tendon-reflex is never really absent, though it may be interfered with by muscular contracture. A slight pseudo-clonus may be observed, but a persistent series of contractions and relaxations are practically never met with unless in contracture, with extension of the ankle-joint, and in the presence of

organic disease ; Babinski's sign is absent. The incompleteness of the paraplegia may in some instances produce modifications in the gait suggestive of ataxia of the cerebellar type or a spastic condition without real rigidity may be present, in which in progression the patient does not use his toes, but walks on his heels ; and Mayer relates that one of his patients could run though unable to walk, and that another could walk backwards more easily than forwards.

Hemiplegia when present, like all the sensory symptoms, (1) is much more frequent on the left side ; (2) the face and tongue are never affected, save in rare cases by spasm in the muscles of the other side ; (3) the muscular power is only partially lost ; (4) usually the arm suffers less than the leg ; (5) muscular rigidity, and (6) Babinski's sign are absent ; (7) the gait is characteristic, the foot being trailed along, and not swung round ; (8) the loss of sensation is usually a marked feature, involving the special senses on the affected side. All these points distinguish hysterical from genuine hemiplegia.

The eyeball-muscles probably never are paralysed, though a pseudo-ptosis is often observed which is the result of spasm of the orbicularis palpebrarum, and paralysis of an ocular muscle is simulated by spasm or over-action in the opposing muscle. Diplopia may occur from spasm in the muscles of accommodation, and may be present when one eye is closed. True nystagmus, seen so often in disseminated sclerosis, never occurs in hysteria.

Spasms and *tremors* resembling those seen in chorea, and constituting the so-called 'hysterical chorea' and chorea magna, are met with ; they are usually more or less rhythmical, of wide range, complex and deliberate in their character, and liable to complete intermissions. A close simulation of genuine chorea may be observed in hysterical patients who have been in contact with choreic patients. Sometimes spasms resembling tetany are observable, but the characteristic features of this disease are always found absent when the signs of Trousseau and Chvostek are looked for.

Vibratory tremors, with rapid or slow oscillations, are common in the seizures or hysterical paroxysms, and may be present in the intervals between them, taking the form of severe clonic spasms ; they are always increased by, if not entirely dependent upon, emotional disturbances.

The intention-tremor of disseminated sclerosis may be simulated, but it is never entirely dependent upon muscular movement, being usually also observable during rest and mental excitement, and there is absence of marked incoördination.

The tremors may affect the head, arms, or legs—in the latter case interfering with progression, and in the former simulating paralysis agitans.

Contractures often follow a fit, but they may appear in patients who have never had a convulsive seizure. The muscles of a limb become the seat of continuous or *tonic spasm*, which fixes the part in a rigid position in which it may remain for long periods till actual structural changes supervene; but this is an extremely rare event. The contracture is always increased in attempts to reduce it by passive movements, and these are painful; it may sometimes last during sleep, but disappears under complete chloroform-anæsthesia.

The upper extremity, when the seat of contracture, is flexed at the elbow- or wrist-joints or adducted at the shoulder; the fingers may be strongly flexed or held in the paralysis agitans position. The lower extremity is usually held firmly in the extended position in all its joints, the heel being very forcibly drawn up, so that the sole of the foot looks directly backwards. The hip- and knee-joints may, however, be the seat of contracture, and, as in all the other joints, pain and hyperæsthesia may be so marked as to closely simulate arthritic disease, and the diagnosis is rendered very difficult by the fact that the joint-contracture may be known to have followed some injury; a close examination will always reveal the fact that the tonic muscular contraction is not limited to the affected joint-muscles, but extends much beyond them, involving other groups, and the hyperæsthesia is widely distributed also. Moreover, the contracture is liable to suddenly vanish spontaneously or to become aggravated by emotional disturbance, and it disappears under complete chloroform-anæsthesia. Contractures are often observed to be distributed in the hemiplegic fashion, and associated with a hemianæsthesia which may affect the entire half of the body or be only observable over the contracted joints or extremities, the trunk and head escaping.

Hysterical trismus is not rare; in this condition, the jaws are firmly closed, or almost closed, so that tetanus is simulated.

Spasm of the neck of the bladder is a common and sometimes painful symptom, necessitating frequent catheterisation, which often causes pain when the instrument is gripped by the sphincter.

Phantom tumours are sometimes observed. These are usually situated in the abdomen about the region of the umbilicus. They are caused by spasm of the diaphragm and irregular paresis of the muscles of the abdominal wall. At the same time gas is imprisoned in a large knuckle or coil of intestine, which is pushed forwards. Such swellings have constantly been mistaken for ovarian and other tumours. In one case, for years under the writer's observation, a mass as big as a large cocoanut existed; it disappeared at rare intervals for short periods, to return again, and totally vanished

once under chloroform after a diagnosis of ovarian cyst had been confidently pronounced by an hospital surgeon. It is stated that upon the disappearance of the tumour under chloroform the liver-dulness may be observed to rise as the diaphragm relaxes. More rarely a phantom tumour has been observed in the centre of a mass of muscle like the calf of the leg.

Visceral symptoms are common, and all forms of gastric disturbances from mere capriciousness of appetite to anorexia nervosa of the gravest type may be met with. It is in this latter condition that the greatest attempts at fraud are observed. Vomiting and gastric pain may simulate cancer or ulcer, and the entire list of the gastric neuroses may be observed. The vomiting of hysteria may be very severe, and the contents of the intestines and even fluids injected into the rectum have been detected in the vomit.

The urine under emotional disturbance is generally greatly augmented, pale or colourless, and of low s.g., and often might be mistaken for water. The opposite condition is occasionally seen, and in rare cases total suppression of urine for days has been observed—a condition likely to occur in cases of severe continuous vomiting.

The heart and respiration may be greatly accelerated ; the attacks of extremely rapid breathing and palpitation usually come on in paroxysms and often after emotional excitement. Asphyxia may be threatened by laryngeal spasm, which may cause cyanosis and alarming stridor. Hysterical coughing, sneezing, and hiccough are sometimes marked symptoms and may last for months, and may be associated with very remarkable sound-productions even when aphonia is present.

The *globus hystericus* is a purely subjective symptom, which may possibly have its origin in spasm. The patient complains of a feeling as if a ball were forced upwards from the stomach or pelvis into the throat, causing distension and producing a dread of suffocation and difficulty or impossibility of swallowing. The globus may be the first symptom of a general fit or convulsion, and often seems like the aura or warning experienced by epileptics. It is in many subjects produced by even slight emotional disturbances.

Vaso-motor symptoms are usually best seen in their local manifestations in association with paralysed limbs or joints the seat of contracture, where there may be a solid œdema of the limb associated with deep lividity. Pallor or deep flushing of the skin, or constant blushing under trivial excitement, is common. Of more interest are the local hæmorrhages which sometimes occur under the skin, and which have led to frauds of all kinds, in which stigmata on the hands, feet, side, and forehead have been artificially produced in imitation of crucifixion-marks. Subcutaneous extravasations of blood do occur spontaneously in rare cases, and bleeding from the

stomach and uterus is not unknown; pseudo-hæmoptysis is common, and is the result of sucking or scratching the gums in order to elicit sympathy. Many skin-eruptions of hysterical origin have been described, some of which are undoubtedly the result of the fraudulent application of irritants; but multiple gangrenous patches on the skin in the vicinity of paralysed limbs, and urticaria are not uncommon. Local sweating is occasionally observed.

Hysterical fever does really occur, but with exceeding rarity, and fraud should always be suspected; the great majority of the hyperpyretic temperatures are the result of friction or pressure on the thermometer-bulb. The body-heat in ordinary hysteria is usually normal throughout.

Symptoms of the Convulsive Fit.—Two forms of convulsive seizure are met with, which differ from each other in the intensity of the symptoms. The *minor* form of hysterical fit is the one commonly seen amongst the members of the Anglo-Saxon race; like the *major* fit, it is liable to endless modifications in the details of the clinical picture.

The patient, usually after some emotional excitement, in which laughter and tears have alternated, is seized with a painful sensation in the throat—the globus—as if a globular body travelling upwards from the pelvis or stomach had become lodged in the throat. There is always a manifestation of fear and sometimes of a distinct aura (as in epilepsy) which may be independent of the globus, the breathing gets quick and irregular, and the patient falls not with the abrupt suddenness seen in epilepsy, but she sinks down with some suspicion of deliberation, sliding into a chair or throwing herself upon a bed, sofa, or other convenient article of furniture, and her body passes through the varied phases of a mild hysterical convulsion. The limbs may at first show some tonic rigidity, the legs being extended stiffly and the arms held at right-angles to the body, but not to the extent seen in the major attacks; and soon, or almost immediately, a series of quick irregular clonic spasms supervene, which rarely present the explosive shock-like character of true epileptic convulsions. The orbicularis palpebrarum, in slight cases, may be the only seat of these spasms, in which case a series of blinking movements, with quivering of the eyeballs, may be alone noticed; but sometimes the limbs are all more or less affected with clonic spasms, which may rapidly pass into purposive co-ordinated movements. These cause the arms, legs, and head to be tossed about or swayed from side to side in a rhythmical way, and the head may sometimes be hammered against the ground, the lips may be bitten, but the tongue never.

Consciousness is not entirely lost in these attacks, though to the bystanders the patient seems completely unconscious, and after the seizure she has some recollection of what has

occurred, and may even be able to repeat observations made by sympathetic friends during the convulsion. Occasionally there is some rambling or incoherent utterance, and evidence of the mind being under hallucinations of fear or terror. Sensation may be somewhat blunted; the pupillary and conjunctival reflexes are present, but they may be slightly diminished; the eyelids are usually closed and tremulous, and resist elevation. The paroxysm may last for several minutes and suddenly cease with some emotional manifestation, or it may last with intermissions for hours, the patient passing into a lethargic or cataleptic condition, with normal temperature. During the apparently unconscious stage in a severe minor fit, pressure upon the ovarian hystero-genetic zone may cut short the attack; firm pressure on the margin of the supra-orbital arch will often produce the same effect, as will also a liberal douche of cold water dashed suddenly and forcibly against the face. Few attacks will withstand a forcible closure of the mouth, if at the same time both nostrils be firmly pinched by the other hand of the attendant; and if a strong induced current be applied to any part of the body a speedy cessation of the convulsions usually occurs.

After the patient has regained complete control of herself and all evidence of the fit has passed away, many of the sensory and some motor symptoms, previously described, may be discovered on careful examination. Usually after the attacks there is a copious discharge of pale urine, and the abdomen may be greatly distended with air, which the writer believes has been swallowed or sucked down during the struggling stage.

Many minor seizures are observed in which consciousness is entirely unaffected, the only manifestations being the presence of the globus, marked loss of self-control, as seen in laughter, crying, and screaming or biting, and sometimes only 'swooning' without any real convulsive movements being observable. Incomplete attacks are common, in which only an incomplete aura is felt, and the only evidence of such seizures may be a sensation of vertigo, which may be mistaken for *petit mal*.

Major hysteria or *grand* attacks, described by Charcot and his school under the title of *Hystero-epilepsy*, are seldom outside of France met with of the degree of severity and with all the characteristic stages or phases described by French writers. The most severe and typical attack witnessed by the writer was in a young robust male subject.

The typical attack may or may not have a *premonitory* stage, with *hysterical* aura, globus, tinnitus, palpitation, and a sensation of fear or terror, or hallucinations. The seizure itself is described as possessing four characteristic stages:

. The *epileptoid* period is ushered in with symptoms so closely resembling epilepsy that a diagnosis made at the moment may be incorrect. There is strong general tonic spasm affecting the trunk and limbs, which are set in a tetanic immobility and which may assume the opisthotonic form ; the eyeballs are turned upwards, the teeth are firmly closed, but the tongue never gets caught between ; frothing of the mouth may occur. The breathing is arrested, and the face, from being pale, becomes congested; a series of rapid clonic spasms follow, with stertorous breathing ; and the entire period, which is sometimes longer than an attack of true epilepsy, passes into a coma of very brief duration.

Consciousness is much diminished, but rarely is it *entirely* lost ; the patient, however, remembers little or nothing of the fit. The convulsions may be arrested by faradism or strong pressure on the ovarian zone, and there is never expulsion of bladder or bowel contents.

2. The *clownism* period soon follows, in which the body assumes a series of grotesque attitudes, the most striking being that described as the *arc en cercle*, in which the patient rests the weight of the body on the *top* of the head and *soles* of the feet, the entire frame being violently bent upwards like a bow ; alternating contortions of various types rapidly follow, often associated with screaming or other emotional manifestations.

3. The *passionate* period or stage of emotional attitudes (*attitudes passionelles*) follows, in which the patient attitudinises in some stage-posture indicative of grief, joy, amativeness, &c., according to the predominating hallucination.

4. The *delirious* period supervenes as consciousness begins to reappear, and visual hallucinations of disagreeable and terrifying objects are demonstrated by the rapid disjointed references made to them in her rambling delirious ravings—a condition which has been named *zoopsia*. Sometimes the patient may by her language show that she fancies she hears voices, and she may carry on a conversation with some invisible person.

The duration of the complete attack from the beginning of the epileptoid period, which lasts but a few minutes (rarely as long as five), to the end of the delirious stage, may not exceed twenty minutes.

At the termination of the fit, the expulsion of flatus from the stomach or the rumbling of gas in the bowel and the voluntary discharge of a copious amount of pale urine may be noticed. There is, as already stated, little or no memory of the epileptoid period ; but during the final stage she may make statements and relate events which have no foundation save in her disordered imagination, and the memory of these hallucinations may often permanently remain as if they were realities ; hence

one of the difficulties in drawing the line between hysteria and insanity. Ormerod insists upon the recognition of the hysterical performances being dictated by some delusion or emotional reminiscence ; this distinguishes them from the automatism of epilepsy, which are repetitions of the trivial acts of daily life or, as in rare cases, the outcome of some unreasoning fury which leaves no memory of its sensations or acts behind it. It is still a doubtful problem to some observers whether the first stage of hysteria major should be regarded as an attack of epilepsy, and the subsequent phases as post-epileptic hysteria. The weight of opinion is with the French authorities who maintain that the attack from beginning to end is purely hysterical; hence the desirability of abandoning Charcot's name, 'hystero-epilepsy,' which he applied to this type of hysteria.

In some cases the attacks are repeated daily, or oftener, and they may pass into a condition known as the 'status hystericus,' in which the patient may remain for days; but this condition is easily differentiated from the status epilepticus, with its high temperature and grave danger to life. At times the patient may pass into a state of lethargy, catalepsy, hypnotism or double consciousness, somnambulism, or ecstasy ; and it must also be remembered that all these states may in rare instances follow incomplete attacks or seizures of the minor type. The cataleptic condition is described under its own title; it may in rare cases of hysterical origin appear as an isolated symptom.

Lethargy, or trance, may likewise make its appearance as an isolated phenomenon, deep sleep seizing the patient suddenly ; but the somnolence is more apparent than real, and, though the muscles are generally relaxed, contractures may be observed, the jaws being often found to be firmly closed. The eyelids resist efforts to expose the cornea, and they often twitch or quiver ; the light-reflex is not lost, but the skin-reflexes disappear though the patellar response is always present. Respiration and heart are usually much slowed, and the cardiac sounds may be so feeble as to be scarcely audible for short periods. The attack may be cut short by pressure on a hystero-genetic zone, or the patient may suddenly or gradually awake, take food, and pass off again into apparent deep sleep. The attack may last for weeks, and there is no memory of this period left in the patient's mind, whilst in the cataleptic condition consciousness may be intact.

Somnambulism occasionally follows a convulsive seizure or appears to take the place of either of the last two periods of the major convulsion. The consideration of this state opens up very difficult problems in psychology which cannot be here discussed; but the condition as witnessed in some rare

cases of hysteria appears to closely resemble, if not to be identical with, the hypnotic state. The patient, though apparently asleep, may be capable of maintaining a conversation, and is able to perform a series of the most intricate and complicated voluntary actions, such as unlocking doors, climbing over obstacles, &c. When consciousness returns, the patient has usually complete amnesia as regards what occurred in the attack, but in a succeeding somnambulistic fit he can remember all that occurred in the previous ones—a condition which may also exist in subjects who have shown no manifestations of hysteria.

Mayer states that the patient's whole bearing and nature show that he is acting under the influence of a delirium which separates him entirely from the outer world, and that he may continue to appear in his 'second nature,' showing an altered character or disposition for weeks afterwards. The exhibition of one side of his dual personality may be readily effected in the hysterical subject by any of the usual hypnotic methods, as gazing on bright light, &c.; and many maintain that hypnotisation is essentially an hysterical or hysteroid manifestation.

Pathology.—There is obviously no pathological anatomy in a disease which is universally regarded as of purely functional nature; whatever molecular derangement may exist in the nerve-centres, it is beyond the range of demonstration by the microscope. Though it is essentially a psychosis originating in disturbed cerebral function, the part played by the secondary derangement of the lower centres, as insisted upon by Gowers, is the keynote to the mechanism which produces many of the symptoms. The nature of the primary disturbance, whether the result of vaso-motor spasm or paralysis, or of an altered state of the nutrition of the nerve-cells, in the present state of our knowledge is mere speculation. Equally unsatisfactory are all the attempts so ably summarised by Ormerod which approach the problem from the psychological side. Janet's view of the identity of hysteria with the somnambulistic state, and its relation to dual personality or to the presence of a higher and lower personal consciousness, may explain some of the phenomena of the major attacks, but it fails to throw light upon the visceral and most of the local forms of hysteria.

Diagnosis.—Many of the points in the differential diagnosis of hysteria have already been emphasised in describing the attacks and local symptoms. The recognition of the disease is very often made in a single glance. At other times it may be the most difficult problem in the entire range of diagnosis, since, as a general rule, it can only be arrived at by a process of exclusion, and such process demands a familiarity with the signs and symptoms of all the organic nerve and brain

affections. This is not all, because hysterical symptoms may be grafted upon those of some serious organic lesion. To treat an organic brain or spinal disease as one of simple hysteria is obviously a more serious error than to regard the functional affection as an organic malady. The writer once witnessed an able disciple of Charcot, after examining and investigating a case of severe headache in a very hysterical woman, confidently affirm that the case was one of simple hysteria within an hour of the patient's tragic death in cerebral convulsions. Probably an ophthalmoscopic examination would have demonstrated the organic nature of the lesion. This is mentioned to emphasise the great importance of searching for any physical sign of organic disease before arriving at a conclusion in obscure cases.

The investigation of the state of the fundus is of primary importance in most cases, optic neuritis or atrophy affording a means of identifying such conditions as *cerebral tumour*, *hydrocephalus*, *abscess*, *meningitis*, or *disseminated sclerosis*. Unmistakable nystagmus and genuine paralysis of one or more ocular muscles, the absence of the pupillary reflex and the presence of persistent hemianopsia, would at once place the case in the organic category, as would also a unilateral paralysis of the larynx.

An unmistakable reaction of degeneration in muscles supplied by a particular nerve in a case of comparatively recent origin, the total absence of the patellar reflex, the presence of Babinski's sign, and of an unmistakable ankle-clonus where there is no ankle-joint contracture, and of *marked* muscular wasting, are certain to be due to organic spinal disease, though some symptoms of hysteria may be also present, and the importance of these signs is augmented if there be incontinence of urine and fæces and a tendency towards the formation of bed-sores.

Paralysis of muscles supplied by a single nerve is almost certainly not hysterical, whilst paralysis of one limb or of a portion of a limb, the other limb on the same side being unaffected, is equally certain to be hysterical.

One of the most characteristic features in all motor and sensory signs and symptoms in hysteria is their liability to continual variations. This character is quite as valuable as a diagnostic sign, as is the close association of them with emotional states.

Disseminated cerebro-spinal sclerosis is perhaps the organic nerve-disease which is most likely to be mistaken for hysteria. The differential diagnosis is given upon page 312, the principal points being the atrophy of the disc, intention-tremor, scanning speech, Babinski's sign, marked nystagmus, and, at a late stage, incontinence of urine. It must always be remembered that this nerve-lesion sometimes appears to belie its organic

nature by a marked arrest or even temporary improvement in all the symptoms. The writer has watched the symptoms of latent hysteria in the sister of a patient who had died of disseminated sclerosis several years before. The disease was accurately mimicked, the symptoms being apparently reproduced without the consciousness of the patient, and a positive diagnosis was impossible for a long period. No suspicion of malingering could be entertained in this patient's case.

Epilepsy is differentiated by the points enumerated upon page 393, and emphasised in the description of the major hysterical seizure on a previous page. *Chorea*, in its relation to hysteria, has been referred to in the description of the spasms.

Neurasthenia should not be confounded with hysteria, though undoubtedly the two affections may occur together. Neurasthenia is more common in middle-life and in males ; the symptoms are nearly all *subjective*, in this respect closely simulating hypochondriasis, the mental depression being much deeper than that usually met with in hysteria. There is almost always a distinct history of some mental overwork or prolonged anxiety. The loss of energy is for the time but slightly influenced by daily variations ; emotional disturbances may be entirely absent, or play but a minor *rôle*, though headaches, vertigo, tinnitus, and a sense of cerebral oppression may be constant.

Joint-disease is so accurately mimicked by hysteria that the physician should be on his guard. The differential diagnosis has been already forestalled in the description of the contractures. The principal points to be remembered are the suddenness of the onset of the contracture, its variability from day to day associated with changes in the emotional state, and the exaggerated degree of the flexion. The most important feature, however, is its disappearance under complete chloroform-narcosis, even when it has been known to exist during natural sleep.

Paralysis of the true hemiplegic and paraplegic types is differentiated by the features already mentioned under the description of the motor symptoms. The greatest difficulty will usually be met with in the elimination of the spastic condition which follows a *transverse dorsal myelitis*. In this affection the presence of Babinski's sign is of great value—*i.e.* the great toe is *extended* instead of being *flexed* by tickling the skin of the sole of the foot, and there is a definite and prolonged true ankle-clonus. The upper border of anæsthesia corresponds to the level of the spinal cord, which is involved in the inflammatory action. The sphincters lose their power, urine and fæces being involuntarily voided, and there is a marked tendency to cystitis and bed-sores.

Primary spastic paraplegia, independent of myelitis and disseminated sclerosis, results from degeneration of the pyramidal tract, or of the posterior part of the lateral tract. The symptoms are similar to those just mentioned, but the sphincters are intact usually and there is no anæsthesia. From hysteria the diagnosis is made by the presence of the excessive reflexes, a true ankle-clonus, and Babinski's sign.

Amyotrophic lateral sclerosis, which may be regarded as identical with progressive muscular atrophy, may be differentiated from hysteria by the above signs of increase in the reflexes, which are also evident in the upper extremities, and by the presence of marked wasting in the muscles. With this wasting is associated contracture of the joints, as seen in hysteria, and the fingers are strongly flexed on the palm. The advent of symptoms of bulbar paralysis also eliminates hysteria without difficulty.

A phenomenon known as '*transference*,' and discovered by Charcot, may be in rare cases of some help in diagnosis. Thus the anæsthesia may be made to disappear from one side of the body to the other by the application of counter-irritants, by electricity, or by contact or approximation of discs of certain metals or magnets—agents which probably act through the mind by suggestion. Hypnotisation has been also recommended by some authorities as a diagnostic aid, the condition being rapidly and easily, as a rule, induced in hysterical patients.

Insanity in hysteria has already been incidentally mentioned. The line of demarcation in some cases between genuine insanity and hysteria may be very difficult to draw. The conditions may undoubtedly occur together, arising out of a marked hereditary neurotic taint. Some hysterical patients certainly become insane, and a small percentage of lunatics may develop or manifest unmistakable symptoms of hysteria ; but the two affections are fundamentally different, even though they may alternate with each other. The relationships of insanity and hysteria are obvious in those cases where one member of a family becomes insane and another exhibits the typical emotional instability characteristic of hysteria. A fact of great importance must not be lost sight of in such cases: the hysterical symptoms may be the first indication of the graver neurosis. An equally important fact is that the delirium and hallucinations of the major attacks constitute a true *transient* mania, almost identical with that observed in some cases of post-epileptic delusion ; but such a mental condition may be considered in a general sense as comparable with the transient delirium or hallucinations which occur in other diseased conditions, as in fevers, in which the memory of the delusions may long remain. As already mentioned, there is no recollection of the post-epileptic

automatic actions left in the mind of the epileptic patient, whilst the hysterical patient may remember her hallucinations more or less vividly after a major attack.

Prognosis. — As regards a decision upon the length or duration of the symptoms there is always great difficulty; the symptoms may suddenly cease in any case, and in rare examples of the disease they may never return. The chances of such a happy result will diminish in proportion to the previous duration of the affection and to the number of recurrent convulsive seizures. The most apparently satisfactory cures may be but transient, the state of the nervous system being in many cases permanent and unalterable; and any serious emotional excitement or grave mental worry may provoke convulsive attacks, especially in those with a bad history of hereditary neurotic taint. The prognosis is best in those cases where the patient's environment can be entirely altered, and many cases are constantly met with in which rapid and permanent recovery has resulted from a change of fortune which has thrown the pampered self-indulged patient upon her own resources in fighting the battle of life.

A carriage-accident, or some startling fright, has not rarely been the means of convincing the hysterical paraplegic patient that she had the full power of her limbs. The outlook is usually bad where contractures of long standing are present.

The disease so very seldom proves fatal that a confident prognosis as regards danger to life may be safely made. Continental authorities describe a rare form of acute lethal hysteria; in this country the few recorded fatal cases owed their termination to laryngeal spasm or to the exhaustion following vomiting. It must not be forgotten that occasionally the transient delirium or hallucinations of the last period of the grand seizures may pass into insanity, but in such cases the graver neurosis was previously present in a latent form. Male patients in whom hysteria is blended with hypochondriasis often gravitate to the goal of insanity.

Treatment.—*Prophylaxis.*—Sometimes it may be absolutely necessary to insist upon the removal of children from their mother when the latter is markedly hysterical and the children are beginning to show some symptoms of being affected with the neurotic taint. Removal to a good school at a distance, under the supervision of a suitable teacher, is often followed by the happiest results.

So, also, when hysteria manifests itself under other conditions, the object of the physician should be to discover the exciting cause and have the environment of the patient altered as completely as possible. The basis of all treatment, to be successful, must be established upon the principle

of placing the patient under a system in which judicious moral control will be firmly and kindly administered. In all severe examples of the disease a cure is impossible as long as the patient is surrounded by her sympathising relatives and friends.

The Weir-Mitchell treatment affords the best hope in such cases. The patient is removed from her home-surroundings and placed under the care of a wise physician and skilled nurse. She is put to bed, and a thorough course of massage with electricity is commenced; food, in an easily digestible form, is administered in liberal amounts at short intervals. The result is generally very striking, and sometimes borders upon the marvellous in those cases of emaciated bed-ridden patients who have long been paraplegic and have suffered from anorexia. The success of the method largely or entirely depends upon the strength of character possessed by the physician, whose firmness and deep interest in the patient's recovery soon secures her complete confidence and absolute faith. This method, unfortunately, is of little value in the well-nourished hysteric patients with good appetites, who from analysis of their sensations and discomforts have become more or less hypochondriacal.

The best plan of carrying out the Weir-Mitchell treatment is to place the patient in a private hospital or home specially devoted to the carrying out of the details of the method. The isolation must be rigid and complete; in some instances it may be necessary to forbid all visits of friends, and even the reception of letters must be prohibited. Forced feeding is seldom necessary, but it may have to be resorted to in some cases. The details of this special form of treatment will be found in the author's 'Dictionary of Treatment,' pp. 437–446.

The use of drugs in hysteria is of minor importance; they are, as a rule, worthless unless associated with moral treatment. It is needless, however, to emphasise the importance of treatment by suitable remedies directed to the correction of such complications as anæmia, chlorosis, dyspepsia, metritis, constipation, &c. Drugging with tonics often does harm, and it is an undoubted fact that many of the symptoms of the disease are made worse by the administration of strychnine, even in very small doses. The class of remedies known as antispasmodics may be used; chief of these are asafetida and valerian. By some authorities these agents are regarded as part of the moral treatment, the patient being compelled to swallow a nauseous and most disagreeable medicine. The experience of every physician proves that many hysterical patients seem to enjoy these disagreeable drugs, and they will continue to take them even without the command of the attendant or nurse.

Such remedies are sometimes of unquestionable value, and their action must have another explanation than that afforded by the theory of their moral influence. All that can be said of them is that they possess some tonic influence over the unstable nerve-centres, and by their administration spasmodic seizures or convulsive attacks are prevented or modified. The best routine agent is 3 or 4 grs. valerianate of zinc, made into a pill with extract of valerian root. It is needless to say that all narcotics must be avoided. The injection of brain-tissue, cerebrin, normal saline solutions &c., probably, when of any use, acts by suggestion.

The treatment of the hysterical paroxysm may be carried out, on the lines mentioned upon page 690, by the employment of those agents which cut short the seizure, as ovarian pressure, cold douching, electricity, temporary blocking of the nostrils and mouth, &c. The actual cautery has been recommended, but should not be used, unless, perhaps, in cases of obvious malingering. The physician who does not realise that the worst manifestations of hysteria may be entirely beyond the power of the patient's will generally fails in all treatment. The confidence of the patient in the attendant is entirely lost when the latter discovers to her that he believes she is either malingering or wantonly giving way to every emotional weakness. Scoldings and every form of punishment only aggravate the condition, and may prove as injurious as the injudicious sympathy of friends. Aphonia is speedily removed by the application of the induced current; often it disappears on the introduction of the laryngeal mirror, and its recurrence may sometimes be prevented by a small blister applied to the exterior of the larynx.

Paralysis in the hysterical subject affords a splendid field for the employment of the skilful physician's powers. Often it yields at once to electricity, but it is prone to return unless followed up by persevering education directed to the restoration of the lost will-power of the patient. The striking results of faith-healing and profound mental emotion sometimes demonstrate to the physician how unwise and ineffectual had been his attempts to scold the patient out of her paraplegic weakness.

Contractures respond less satisfactorily; strong or violent attempts to straighten out the flexed joints often do harm, but sometimes good results are obtainable by the induced current. Passive movements combined with massage should be persevered with, and these also are of great value in the treatment of paralysed limbs. Sometimes the knowledge that the contracture has entirely yielded to chloroform has a profound effect upon the patient after she has recovered from the anæsthetic. Blistering of the contracted joint is of much value, but it has the drawback of interfering with passive

motion and massage. As in the treatment of paralysed limbs, the passive movements should soon be supplemented by voluntary exercises, the patient's will-power being slowly and perseveringly educated day by day. Without judicious moral treatment directed to the general condition or psychosis, all the local symptoms of hysteria are prone to recur after their disappearance has been effected by the measures described, and not uncommonly new local symptoms crop up.

Anæsthesia is liable to disappear if left severely alone; experiments in transference by metals and magnets are liable to perpetuate it.

Hyperæsthesia is best relieved by counter-irritation. Small blisters applied to the regions in which neuralgic pains are experienced are often efficacious; but their use should not be continued for any lengthened periods, otherwise the patient is led to concentrate her attention too closely upon the affected zone, and it may even be sometimes necessary to apply smart counter-irritation at a considerable distance from the painful spot.

Affections of the special senses as a rule require no treatment beyond the use of the general moral and antispasmodic agents already referred to. Vomiting and severe anorexia are pressing indications for the Weir-Mitchell treatment; both are sometimes speedily cured by forced feeding by means of the stomach syphon or pump, the food introduced in this way being often retained whilst that which is swallowed is rapidly ejected. Rarely is rectal feeding of much value.

After the removal or amelioration of the local and constitutional symptoms, every effort should be made to change the environment under whose influence the psychosis had been fostered; and above every other consideration is to be placed that of procuring some occupation or work—mechanical, domestic, or philanthropic—which will give to the hysterical subject a new interest in life.

ICHTHYOSIS

This is a congenital and very frequently an hereditary affection of the skin, characterised by the presence of a thick coating of epidermic scales associated with great dryness, and resembling in some instances the scaly appearance of fishes; hence its name, and the English synonym of 'Fish-skin Disease.'

Etiology.—In the majority of instances either a distinct hereditary history is obtainable, or else several members of the same family are found to be affected; boys suffer as frequently as girls, but sometimes in a family only the children of the same sex may be affected. It has been said to appear in

the offspring of parents suffering from psoriasis, but the occurrences are probably to be explained as accidental coincidences, since the diseases are quite distinct. An acquired condition of the skin resembling ichthyosis has been observed to occur in connection with peripheral neuritis and some nerve-diseases, but it is essentially different. All that can be said of the causation and pathology of ichthyosis may be summed up in the statement that it is an error in development.

Symptoms.—The disease presents itself in very varying degrees of intensity, and is usually first noticed a few weeks after birth. In mild cases the surface of the skin is covered over with small dry conical elevations, which give the sensation, as the hand is passed over the affected skin, as if it were feeling a nutmeg-grater. These projections are caused by the prominence of the hair-follicles, and are best seen and felt over the extensor aspects of the limb. Some general thickening of the epidermis is usually perceptible, and there is almost entire absence of perspiration ; such cases are usually described as *Xerodermia* or *Keratosis pilaris.*

In the more marked cases known as *Ichthyosis simplex*, or *I. nitida*, the entire cutaneous surface is covered over with dry scales, varying in size from the powdery flakes seen in dandriff of the scalp to large plaques of polygonal area which are suggestive of the hide of the crocodile or rhinoceros. The scales vary in colour from a dirty green to a dark brown, owing to admixture of dirt and decomposing sebaceous matter, though the oily as well as the watery secretion of the skin seems to be almost absent. Though the affection is general and symmetrical, some parts of the skin suffer much more than others. Thus the face is but slightly or not at all affected ; the soles and palms usually escape with the arm-pits, groins, genitals, front of the elbows, and popliteal spaces ; whilst the extensor regions, especially the knees and elbows, may be encased in thick, dense, tesselated plaques.

The hair is scanty, dry, and friable, and the nails are brittle but not deformed.

The diseased skin is very liable to become affected with eczema and is very sensitive to changes of temperature ; the condition is always more or less relieved by the heat of summer and aggravated during the prevalence of cold winds.

The general health suffers little, though sometimes there may be considerable itching ; and whilst improvement can be effected by judicious treatment, the condition, which usually reaches its height before puberty, is incurable.

Pathological Anatomy.—The essential change consists in a greatly increased production of epithelial cells, which soon become keratinised. The deeper layers of the skin are involved to some extent in all severe cases, large round or spindle cells being found in the cutis ; but these changes are probably

secondary to the hyperkeratosis of the horny layer, which is always thickened considerably and possesses an abnormal amount of cement-substance. There is wasting of the hair and sebaceous follicles, though little change is noticed in the sweat-glands.

Ichthyosis hystrix, or porcupine-skin, is a congenital affection involving *localised* areas of the skin ; these are affected with warty growths capped by thick plates of brownish-green horny epidermis of polygonal or mosaic outline, which project above the level of the skin, resembling the severe forms of simple ichthyosis. The patches are generally unilateral in distribution, following the direction of the cutaneous nerve-trunks—*i.e.* taking a course parallel with the long axis of a limb or a transverse direction on the trunk. As the horny masses become detached, the underlying warty growths or hypertrophied papillæ, which are the main pathological feature in association with the hypertrophy of the horny layer, become visible. These often bleed freely and may ulcerate.

Harlequin fœtus is another form of ichthyosis in which the prematurely born infant is covered over with a layer of thickened epidermis which is divided into sections or areas of square or polygonal outline by fissures, causing a resemblance to the variegated garment of the harlequin. The disease is only of pathological interest, being usually associated with other abnormalities, and it is always fatal. It is supposed to be the manifestation of an intra-uterine ichthyosis, several cases being reported of an attenuated form, which ultimately passed into the condition of ordinary congenital ichthyosis, though a few such have been known finally to end in recovery.

Ichthyosis of the tongue, so-called, has no connection with the above diseases, in all of which the mucous membranes escape. The condition will be referred to under ' Leukoplakia.'

Treatment.—Drugs by the mouth are generally useless in all the forms of the disease, but Pringle reports much benefit from the administration of thyroid extract in some cases. Local improvement can only be expected to follow the continuous application of lanolin or oil after the removal of the hypertrophied epidermis by prolonged immersion in warm weak alkaline baths, followed by gentle friction. Recently Jackson claims to have cured a case of ichthyosis by thorough irrigation of the colon—a method based upon the hypothesis that the hyperpigmentation and hyperkeratosis are the results of hepatic and intestinal disturbances.

In I. hystrix the isolated growths may be disintegrated by a paste of salicylic acid, or they may be removed by the curette, or by the knife or galvano-cautery under local anæsthesia.

ICTERUS. (See JAUNDICE.

IMPETIGO CONTAGIOSA

This is the name given to an affection of the skin characterised by the presence of a pustular eruption caused by inoculation with staphylococci.

Etiology and Pathology.—It is still maintained by a few dermatologists that a true pustular eruption may occur which has no contagious or infective properties, and hence the old simple name of Impetigo is still retained; but the evidence afforded by bacteriological examination leaves little, if any, room for doubt that every case of impetigo is the direct result of inoculation with pus cocci, chief amongst which are staphylococci. Streptococci probably may give rise to pustulation identical with that caused by staphylococci. The disease has been traced by Payne to its origin in such suppurative conditions as ulcerative stomatitis, conjunctivitis, and otorrhœa, and he has found these affections, conversely, originating from the infective matter of the pustules of impetigo.

Impetigo is liable to occur in epidemics, and affects children, especially ill-fed and neglected ones, much more frequently than adults. Scrofulous subjects are very susceptible, but the eruption may be found upon the healthiest. The staphylococci which cause impetigo are constantly found on the normal skin as well as in boils and carbuncles, and some factor must be operative which increases their virulence in order that impetigo may be produced; this Payne supposes to be the passage of the cocci from one focus of inflammation, or from one individual, to another. Thus the cocci are very prone to attack some injured portion of the skin, after which their virulence is always augmented; and their activity further becomes intensified by transplantation upon abrasions on other subjects. This is the explanation of the very severe form of the disease known as *Football impetigo*, which occurs in the epidemic form.

Inoculation is usually accomplished by the finger-nails, which plant the micro-organisms in the scratches made to relieve itching, but the pustules may spread over the surface, independent of abrasions.

Symptoms.—The eruption, if seen early, may be found to be vesicular, but it so rapidly becomes pustular that many believe in its pustular form from the very beginning. Isolated vesicles or pustules soon combine into clumps or groups; they burst and discharge a syrupy fluid, which rapidly dries up, forming yellow crusts or scabs. These are not surrounded

by any red areolar blush, and the skin is quite normal between one cluster of the crusts and its neighbour. Occasionally bullæ may appear whose contents become pustular before bursting, but as a rule these occur only when the disease attacks adults, and when the hands of cachectic children are the site of the eruption.

When the scabs or crusts are removed a raw surface is exposed, from which a thick fluid oozes which dries into a new crust. The scabs, if left alone, will be shed, leaving no mark but a reddish stain, which soon disappears.

The parts of the body which are usually exposed suffer most, and the face is more frequently attacked than any other part, the chin being a favourite site. The entire face in rare cases may be covered with coalescing crusts which form a mask of considerable extent. The lymphatic glands in the neighbourhood become enlarged and tender almost as soon as the crusts appear ; the superficial glands below the jaw and over the occiput are commonly involved, and in very cachectic children they may suppurate. In the epidemic forms of impetigo the pustules may be found scattered irregularly over the entire body, and the eruption usually in these cases runs a definite course, the micro-organisms apparently losing their virulence in about ten days, unless transferred to another individual.

Neither in the sporadic nor epidemic varieties does the eruption ever assume a bilateral or symmetrical type, as in many other skin-affections. In cachectic subjects and in adults, where bullæ are commonly seen, and in regions which are exposed to friction, the scabs may be surrounded by an inflamed areola, and as they drop off an ulcerated surface is left, which may afterwards show scarring. It is this type of the disease which, in unhealthy subjects, has been described as Ecthyma, but there can be no doubt that ecthyma is but a phase of impetigo, being caused by the same micro-organisms.

Eczema impetiginoides, as already described upon page 350, is a condition in which the staphylococcus is planted upon a true eczema of the typical vesicular or weeping type.

Impetigo of the scalp, also called Porrigo, is often obstinate and severe, especially when associated with pediculi, the infective process probably being intensified by the staphylococci finding their way into the superficial layers of the skin through the bites of the insect and the abrasions caused by scratching.

Diagnosis.—The appearance of the yellow crusts, with the healthy state of the skin between, which gives the discrete character to the eruption, at once distinguishes it from the weeping surface of eczema, which is, moreover, often also symmetrical. The pathognomonic test of impetigo is the ease

with which the discharge can be inoculated upon a fresh surface. This also explains how in some cases the disease, under conditions favourable to the growth of the staphylococci, may remain chronic and exist for an indefinite time.

Treatment.—This will consist, in the first instance, in the removal of all scabs or crusts by covering them over with several layers of lint soaked in a saturated solution of boric acid, over which oiled silk is placed. The entire surface of the body should be submitted to the action of a weak antiseptic, and a warm bath with permanganate of potash solution affords the safest means of carrying out this object.

The sores may be dressed with boracic-acid ointment or with zinc ointment to which 5 per cent. of white precipitate of mercury has been added. The affection rapidly yields to cleanliness and the above antiseptic measures ; at the same time the cachexia and any anæmia present should be met with iron and good feeding. Rarely will be it necessary to use remedies to allay itching, as this is never distressing, but 5 or 10 grs. of menthol may be added to each ounce of ointment if necessary.

INDIGESTION. (See DYSPEPSIA.)

INFANTILE PARALYSIS. (See under PARALYSIS.)

INFANTILISM

This condition, as defined by M. Meige, is a form of defective development characterised by the persistence of the physical characters of childhood in one who has reached or passed the age of puberty. The development of the mental faculties as a rule keeps pace with the progress of the physical growth, so that there is more or less mental backwardness, and, as usually stated, the adult remains a child in mind as well as in body. The retarded physical development or bodily defect, as pointed out by J. Thomson, is seen in the arrest of the growth of the *primary* sexual characteristics (a rudimentary condition of the sexual organs), as well as in an absence or rudimentary state of the *secondary* sexual characteristics. These latter are adult proportions of the figure, laryngeal enlargement, facial, pubic, and axillary hair.

Myxœdematous infantilism represents the most common type of infantilism, or, as it is also sometimes called, myxœdematous dwarfism. Byrom Bramwell applies the term 'pathological dwarfism' to this condition, to distinguish it from normal or physiological dwarfism, in which the mental

and the sexual development are perfect, the only departure from the normal state being seen in the deficiency of height. The term 'dwarfism,' he points out, should not be used as a synonym of infantilism. Subjects of infantilism are dwarfs, but a dwarf whose sexual development is normal, must not be regarded as an example of infantilism.

The cause of the myxœdematous type obviously is defective function of the thyroid gland, and the condition is identical with cretinism. The dwarfage in these cases is so characteristic that it is impossible to mistake it for any other form. The genital organs are rudimentary; pubic and axillary hair is absent; the voice is cracked and harsh, and the physiognomy pathognomonic; the physical configuration and mental state remaining in the condition seen in childhood. The changes in the skeleton are due to an arrest of ossification, which shows itself in the delayed union of the epiphyses and diaphyses of the long bones.

Pancreatic infantilism is a type of arrested development or dwarfage, first described by Bramwell as a definite clinical entity due to disease of the pancreas. The condition, pathologically, is closely related to the myxœdematous type, since in both varieties marked improvement follows the administration of preparations of the gland-substance whose absence causes the dwarfage. Diarrhœa was a prominent feature in Bramwell's case and in an example of the disease described by J. Thomson; probably many of the cases of stunted development associated with chronic diarrhœa may belong to this category.

In pancreatic infantilism, unlike what is constantly observed in the myxœdematous type, the mental condition is normal, and there are no evidences of the physical peculiarities always seen in cretinism. Whilst the genital organs exhibit the usual infantile condition, the voice is high-pitched and child-like in tone. J. Thomson and Bramwell also recognise a type of infantilism associated with anæmia and enlargement of the spleen.

Lorrain's type of infantilism consists simply in a diminution in the stature and bulk of the otherwise well-proportioned body, the individual presenting the physical characters of a miniature man or woman. The axillary, facial, and pubic hair are absent or exceedingly scanty; the genital organs, though small, are, according to J. Thomson, well-shaped and quite large enough for the size of the body; and the intelligence is generally normal.

The condition is usually regarded as the result of deficient or defective arterial development, which prevents the proper nourishment of the different tissues and organs; these consequently remain dwarfed or stunted. The causes of this *anangioplasmia* are congenital syphilis, tuberculosis, congenital

malformations of the heart, and probably some forms of toxæmia of unknown origin. Thomson points out an important difference in this form of dwarfage from the myxœdematous type—the diminution in stature is not due to defective ossification, but to its premature occurrence, since the epiphyses are found to be normally united.

Hastings Gilford's type of infantilism has already been referred to under 'Ateleiosis' (page 96). It is described by Bramwell under the title of 'Infantilism without apparent cause,' but this authority does not regard the condition as a definite variety of dwarfism or of infantilism. Two varieties are recognised. In one there is delay of growth and development till the onset of puberty, after which the sexual system matures and the body-development remains permanently arrested. In the second variety there is no maturation of the sexual system, the individual exhibiting both dwarfism and infantilism throughout life, though the growth may continue up to the age of thirty or even later. Both varieties of ateleiosis may begin at any period during the early years of life—*i.e.* either in infancy or early childhood. There is usually a somewhat cretinoid physiognomy, but the intelligence is normal.

ACHONDROPLASIA, or CHONDRODYSTROPHIA FŒTALIS

This is a rare and interesting form of dwarfage. It does not belong to the category of infantilism, since the genital organs are perfectly developed; it is described here under 'Infantilism' for convenience, and for the purpose of contrasting it with the above-mentioned varieties or types of infantilism.

Achondroplasia is characterised by deformities of a remarkable type, entirely different from those seen in other forms of dwarfage. These deformities are the result of defective development of the bones during fœtal life. Only those bones which develop in cartilage are affected, and only those which grow in cartilage during the *early period* of intra-uterine existence are involved in the dystrophy. Thomson points out that the bones which develop from membrane, and the cartilage-formed bones in which ossification does not begin before birth, or not till late in fœtal life, escape, or are but involved to a very slight degree. Hence the long bones of the limbs, the ribs, the posterior part of the base of the skull, and the ossa innominata suffer, whilst the bones of the cranial vault, the clavicles, sternum, and short bones of the carpus and tarsus are unaffected. The developmental error is mainly confined to the cartilages of the epiphyses.

The deformity, though present at birth, attracts little attention till the child grows up. The stature is markedly stunted, so that at the age of thirty the individual may not

measure four feet: usually he measures considerably less. The head looks disproportionally large for the body; it usually is only of average size. The trunk is of normal dimensions, but the arms and legs are much shorter than they should be, often not exceeding half the normal average length. The bones of the arms and legs are thick and strong, showing exaggerations of the normal curves, which still further increase the stunting and deformity. The humerus and femur are shorter than the radius and tibia, the length of the lower limb accounting for the shortness of the stature.

As the subject stands erect, the finger-tips, when the arms hang by the side, reach only to about the upper margin of the great trochanter. The gait is waddling and the physiognomy usually striking; the nose is abruptly depressed at its root; the head is square, and a slight degree of hydrocephalus may be observable. The broad and shortened hands are highly characteristic, exhibiting the 'trident' position when placed flat upon any object, and the fingers are thick and not quite straight. There is usually exaggeration of the lumbar curve, so that lordosis may be apparent.

Both sexual and mental development are normal, so that achondroplasia must not be confused with infantilism. The pathology of the condition is unknown; most authorities regard it as quite distinct from fœtal rickets and cretinism, and it is uninfluenced by thyroid or other treatment.

INFLUENZA

This is an infectious disease, the result of a specific microbe. It breaks out in different parts of the earth simultaneously in great epidemics, which appear from time to time at long and irregular intervals, and its most striking characteristics are the rapidity with which it spreads, the enormous numbers of persons infected by it, the abruptness of its onset, and the shortness of its incubation.

The disease is known by a number of other names, as La Grippe, Russian Influenza, &c., and must not be confounded with the ordinary epidemic catarrh.

The last great pandemic first gave evidence of its presence in the interior of Russia in 1889. Spreading in the autumn to St. Petersburg and Moscow, it rapidly swept over Europe, invading in a few weeks the British Isles, passing over to the American continent, which was involved from north to south; Australia, Africa, and India, and the greater portion of the entire globe were subsequently visited before the expiration of 1890. Since this period the disease has been endemic, and at short intervals minor brief epidemics have been noted in various countries and localities. In the severe

pandemic form, the virulence of the disease exhausts itself usually within two months in each particular district affected. The temperature of the atmosphere has little influence over the spread of the disease: many of the worst epidemics in this country occurred in midwinter.

Etiology.—The telluric or atmospheric conditions which contribute to the periodical virulence of the bacillus of influenza or to the susceptibility of the human subject are practically unknown; but, notwithstanding the marvellous swiftness of its extension over large tracts of the globe, most authorities agree that this is not more rapid than can be accounted for by the ordinary rate of the methods of human intercommunication. The disease is highly contagious and is conveyed readily from those affected, though at the beginning of an epidemic the enormous number of those stricken down with it simultaneously conveys the idea of an entire district being enveloped in an aërial ocean saturated with the disease-germs. At a later stage of the outbreak those who, by reason of greater resisting power, had escaped, finally succumb to the more concentrated infection proceeding from the emanations of those suffering from the disease.

Some instances are recorded of small localities and communities in schools and asylums which escaped entirely during the last great epidemic.

The disease is undoubtedly conveyed in the pulmonary exhalations from the affected, probably by those coming in contact with them inhaling the air mixed with the fine spray of mucus ejected in the act of coughing, since droplets of mucus have been demonstrated containing the specific bacillus.

The endemic or sporadic cases which continue to crop up for some years after the cessation of the greater epidemics are also to be regarded as foci of infection requiring rigid isolation, especially as a previous attack of the disease does not confer immunity; and it has been proved that the buccal, nasal, and bronchial mucus may contain the bacillus for a considerable period after the apparent recovery of the patient.

Pathology and Morbid Anatomy.—The specific microorganism was first recognised by Canon and by Pfeiffer independent of each other, and is now known as Pfeiffer's bacillus. It is a very small bacillus, staining well with carbofuchsin or Loeffler's methylene-blue, the stained protoplasm showing at each end, which causes it to resemble a diplococcus.

It is found in abundance in the nasal and bronchial mucus and in the lymphatics of the air-tubes and pleura, but not in the blood. The toxins manufactured by the bacillus in the lung are absorbed, and, being carried by the blood to the nerve-centres, produce the symptoms of constitutional poisoning which characterise the onset of the disease. Other

organisms are constantly found in association with the specific bacillus, as staphylococci, streptococci, and the diplococcus of pneumonia—a fact which explains the protean manifestations of the disorder. The writer has closely watched the progress of the disease in an outbreak which attacked nearly every child in a comparatively isolated school containing over 200 inmates, not one of whom presented a single bronchial or pulmonary symptom, though such symptoms were the rule in those at the same time suffering in the surrounding districts—a fact which leads him to believe that other micro-organisms are nearly always acting along with the influenzal bacillus.

The pulmonary changes seen in fatal cases are usually those found in acute broncho-pneumonia, with patches here and there apparently identical with those seen in true croupous pneumonia. It is quite possible that both diseases may occur together, and in some cases the *post-mortem* signs are suggestive of a true diplococcic pneumonia being engrafted upon the influenzal condition; whilst in others the changes are indicative of an interstitial inflammatory condition caused by the influenzal bacillus alone, or in conjunction with some other hitherto unrecognised micro-organism. This supposition is strengthened by a study of the physical signs heard over the chest during life in patients who recover, as will be mentioned later on.

Symptoms.—These appear, usually with marked abruptness, after an incubation-period which may be as short as twenty-four hours or as long as five days. The first symptom noticed by the writer in his own person on three separate occasions, and elicited from other patients capable of noticing clearly their sensations, was a marked hyperæsthesia of the skin, chiefly of the region of the trunk on its dorsal and lateral aspects. Almost immediately after this was experienced a free secretion of urine, followed by weakness of the legs, sometimes amounting to transient paraplegia. The hypersensitive state of the skin is rendered obvious to the patient as he leans back in his chair, and may be so marked for several minutes as to simulate the sensation of a mild sinapism or the application of a coarse horsehair shirt. A slight shivering fit, or a smart rigor, often supervenes, and in less than an hour this is followed by severe and generally excruciating backache, with intense frontal headache and pain at the back of the orbits and racking pains in the joints, bones, and muscles. The temperature usually rises rapidly, and may, within a couple of hours from the first symptom, register 103° F. The pulse usually does not rise as rapidly as the fever, but it ultimately becomes quickened; there are thirst and great restlessness, with a remarkable feeling of physical and mental prostration, usually exceeding that met with in the onset of a severe attack of typhus.

The state of the patient at this stage closely resembles that of the victim of an aggravated inflamed or 'sore throat,' without the throat-symptoms. The flabby tremulous tongue is early coated with a thick, white, moist fur; the breath is offensive, the urine becomes scanty, and the skin dry and hot; perspirations appear later. Insomnia is always present, the result of the physical suffering, and some mental aberration or mild degree of delirium is not very unusual. There may be no bronchial or pulmonary symptoms, but in the majority of cases a dry cough, with some secretion from the nasal membrane, appears early.

After as early as thirty-six hours, or as late as three days, the temperature in this simple type of the disease has fallen to normal, though the general aching of the bones may remain longer. Sweating, alternating with slight chills, is often experienced after the second day, and in some cases drenching sweatings may continue to come and go for many days. Even where the temperature has fallen to the normal by the end of twenty-four or thirty-six hours, the patient may feel himself prostrated and depressed in mind and body, as much as if he had passed through a long fever.

Whilst the above brief description covers the majority of the cases of the simple *febrile* type of influenza, it must be said that the symptoms of the disease in the minority are most variable, and sometimes perplexing. Several distinct types of the affection are met with in epidemics, and it is interesting to remember that a particular type may be peculiar to a particular epidemic or pandemic, probably because another micro-organism is then associated with the specific bacillus.

The *catarrhal* type begins with the symptoms already described, but coryza and the other signs of an acute catarrh or 'infectious cold,' if not present from the first, soon make their appearance, and an irritating cough, from catarrh of the larger tubes, adds to the patient's discomfort. The nasal, pharyngeal, and bronchial irritation may spread downwards and end in an acute capillary bronchitis with a profuse tenacious purulent secretion causing dyspnœa and cyanosis which may last for many days or weeks.

The *pulmonary* type may develop out of the simple febrile or catarrhal form, or the case may present the features of a pneumonia from the start. The *pneumonia* of influenza is often fatal, and though it presents considerable complexity and much variability in its signs and symptoms, these characters often are sufficient to distinguish it from the true croupous form, which, upon the whole, varies but little in its manifestations. It will simplify the description of influenzal pneumonia if those cases are excluded in which an apparently typical invasion of the Fränkel's diplococcus occurs in a

patient suffering from influenza; these cases are recognised by the identity of their signs with those of the true croupous type of the disease, and they only occur in a small percentage of the pulmonary type of grippe.

The pneumonia characteristic of influenza is of lobular or catarrhal form in the great majority of instances. It usually begins at the base of one or both lungs behind, without very definite dulness or increase of vocal fremitus or resonance. The auscultatory signs are almost pathognomonic: crepitations of various degrees of fineness and quality are heard at various spots over the affected organ, whilst normal sounds may be audible over the intervening areas of healthy or uninvaded lung. True bronchial or tubular breathing may be heard at one spot, and if the chest-piece of the stethoscope be moved for half an inch to either side of it, fine râles, such as are considered characteristic of the first stage of croupous pneumonia are audible. Another slight shifting of the instrument may reveal the sharp crackling râles of broncho-pneumonia, or the moist bubbling sounds heard in acute bronchitis. Thus, within a space not larger than a crown-piece all the signs characteristic of every stage of pneumonia and bronchitis may be heard. A number of affected islets may coalesce to the extent of producing a patch as broad as the palm of the hand, over which dulness may be distinct, and other signs of consolidation may be obtainable; but if this area be patiently explored, perhaps in its very centre a spot may be discovered in which sharp cracklings are audible, which are well described as sticky râles by Goodhart. The disease tends to spread irregularly over the entire lung, and in not a few instances the writer has watched its slow progress, as it crept from the base to the apex on the posterior aspect and then slowly travelled downwards from apex to base in front.

There is another characteristic feature in influenzal pneumonia which was demonstrated in the last great pandemic in many cases seen by the writer—*it is highly infectious.* This infectiousness of the pneumonia is quite distinct from the infectiousness of the primary influenza; thus patients suffering under, or recovering from, the simple febrile form of influenza, when brought into contact with a pneumonic case, very frequently developed the lung-symptoms, and it was comparatively rare in the writer's experience for a single case of pneumonia to appear in a large household. Even in hospital, during the epidemic, when a pneumonic case was placed beside convalescing influenzal patients, not unfrequently the latter were noticed to contract the lung-complication.

This feature in the contagiousness of the pulmonary type of the disease suggests the idea of a dual infection.

With the progress of the pneumonia the symptoms all deepen: the temperature keeps high, the pulse accelerates, the cough becomes difficult and frequent; the secretion brought up may be very viscid, and blood is often expectorated. The respiration-rate is usually greatly quickened; there is marked deficiency in the aëration of the blood; delirium commonly sets in, and the heart is prone to fail suddenly. If the inflammatory mischief attacks the pleura the effusion is very liable to end in *empyema*. The catarrhal, pulmonary, and pleural types are usually regarded as but phases of one form or type of influenza, usually called the *respiratory* type, as all the symptoms not infrequently are found in the same patient, and, as already stated, they may be present from the first onset, or they may appear after a brief cessation of the fever, when they are usually regarded as a relapse, or as a sequela of the primary disease.

The duration of the disease is indefinitely lengthened by the thoracic complications if the patient does not succumb early to the pneumonia or to cardiac failure.

Gastro-intestinal symptoms are so common in some epidemics as to constitute a distinct *abdominal* type of the disease, in which profuse painful diarrhœa, sometimes associated with vomiting, dangerous collapse, jaundice, splenic enlargement, and delirium occur.

In this form of the disease, as in the next, there may sometimes be but slight fever, or even a sub-normal temperature throughout. The *nervous* type prevails in some epidemics; and in rare cases the symptoms are those of an acute hæmorrhagic encephalitis, setting in suddenly or during the febrile stage, with stupor, which may rapidly pass into coma. In others the symptoms are those of a polio-encephalitis superior, in which the ocular muscles may all be paralysed; cerebellar ataxia and bulbar paralysis may follow, along with spinal or hemiplegic symptoms.

Meningitis is not uncommon, and abscess of the brain may occur independently or as the result of acute middle-ear disease, which is common. Drowsiness may be as prominent a symptom in some cases as insomnia is in others. Mental symptoms may be the predominant feature in the nervous type; commencing as an exaggeration of the depression seen in all forms of the disease, it may develop suddenly into wild delirious mania, or it may steadily deepen into melancholia, or terminate in dementia. Suicidal tendency is extremely common as a post-influenzal phase of melancholy.

Neuritis may appear early; more frequently it occurs as a sequela, and may present itself in many forms, which simulate paraplegia, ataxia, or neuralgia. The symptoms are in the main those of a typical multiple neuritis simulating the clinical picture of alcoholic neuritis, but in some cases

only one or two nerve-trunks may be affected. Loss of sensation may be combined with the weakness in the muscular apparatus, and one of the commonest symptoms is loss of the sense of taste and smell, which in some cases may never return. Symptoms like those seen in Menière's vertigo are not rare.

The cardiac type.—Of the sequelæ, or complications of influenza, none is so serious as the crippling of the heart which so often follows an attack in elderly subjects, but it must not be forgotten that the main feature, from the onset of the disease, may be sudden cardiac failure. This may show itself by an extremely rapid and feeble pulse, some dyspnœa, and a tendency to syncope, which may prove fatal during the first week of the disease. Acute dilatation is liable to occur in hearts which were not previously sound, and this is the explanation of the alarmingly rapid rupture of compensation which is witnessed in subjects who have valvular lesions without symptoms. Bradycardia, tachycardia, and marked errors in the cardiac rhythm are very common symptoms or sequelæ.

Thrombosis of veins from phlebitis, and in some instances malignant endocarditis or pericarditis, are met with. Albuminuria is very common in some epidemics; and post-influenzal diabetes, though rare, has been reported occasionally. All kinds of erythematous rashes: herpes, pemphigus, purpura hæmorrhagica, &c., have been described; but it is hard to resist the temptation of believing that many of these were drug-eruptions caused by the remedies administered, since antipyrin was so freely prescribed. The sub-normal temperature, which remains long after the attack has passed off, cannot, however, be attributed to this drug.

Parotitis has been observed as a complication or sequela, and in a few cases orchitis has been reported alone or in association with it. Eye-troubles, as conjunctivitis, keratitis, iritis, and tedious weakness or insufficiency of the eyeball-muscles, are common.

Of all the complications or sequelæ, none are so common as the prolonged mental prostration which exhibits itself in inability to concentrate the mind upon any work. In some degree this follows every attack in patients of middle-age or later, and resembles the complete mental inertia met with in severe cases of neurasthenia following severe and prolonged cerebral work or excitement. Like the other sequelæ, it may follow an attack of influenza which was so mild and short as almost to have escaped the notice of the patient; and the condition may last for many months, and render him unfit to follow any occupation requiring the least mental exercise. It is very often complicated with facial or other neuralgias, the result of neuritis.

Diagnosis.—When the disease appears in its pandemic form the diagnosis rarely offers a difficulty: the suddenness of the onset, the intensity of back- and head-aches, the racking pains in the joints and limbs, and the profuse sweating at a later stage, are, when taken together, quite distinctive. The only epidemic disease which prostrates its victims so suddenly and in such appalling numbers is *dengue*; but it only flourishes in very hot seasons and in tropical or sub-tropical climates, and there is constantly the transient eruption present which settles the nature of the epidemic in a few days.

Typhoid fever and true *croupous pneumonia* were often overlooked during the great epidemic, the patients being supposed to have influenza; but the shortness of the fever of influenza, with the suddenness and rapidity of its onset, are usually sufficiently clear to prevent mistakes when ordinary care is observed in the physical examination.

A form of influenza, in which recurring chills and profuse sweatings are prominent features, may be mistaken for *malaria* in regions where this disease is prevalent, and in temperate climates it may for some days be regarded as a manifestation of *septicæmia*, *pyæmia*, or *ulcerative endocarditis*. The subsequent progress of the disease may be the only means of eliminating these affections. The recognition of the specific bacillus in the sputum affords a demonstration of the nature of influenza; examination should never be omitted in doubtful cases. It was the only way in which the writer could eliminate *acute phthisis* on several occasions in sporadic cases where the chest-symptoms lingered for weeks.

It must be always remembered that any disease which has remained latent before the onset of influenza may be brought into striking prominence after the specific fever has subsided. This has been already referred to when mentioning the cardiac type of the disease. In the post-influenzal heart pure and simple there will be no evidence of the hypertrophy which is characteristic of old valvular lesions, though a temporary dilatation, with regurgitant murmur, may complicate the diagnosis for a time.

Organic nerve-diseases afford the most complex diagnostic problem, as nearly every form of lesion may be simulated, and a correct diagnosis is often impossible without waiting for a considerable period. The important question always crops up, Are the symptoms the result of a neuritis or poisoning of the nerve-centres, or are they the manifestations of a lesion, previously overlooked, which has been brought into prominence by the influenzal attack? *Disseminated cerebro-spinal sclerosis* has been noticed in several instances to follow influenza, and by some authorities it has been mentioned as a sequela; it is much more likely to have been present in a dormant or latent form before the attack as this perhaps more

frequently than any other organic nerve-disease has a long latent period or remission of all the symptoms. In the same way locomotor ataxia and ataxic paraplegia have been incorrectly ascribed to influenza. *Alcoholic neuritis* resembles the post-influenzal form, but a careful investigation of the patient's past history may supply evidence of the cause and of symptoms which had been present before the influenzal attack. On the other hand, a post-influenzal neuritis may be mistaken for an organic nerve-lesion, the attack of influenza being so slight as to be forgotten or overlooked by the patient; such sequelæ often follow the mildest seizures, as is also well recognised in the case of diphtheria.

Prognosis.—Considering the enormous numbers of patients stricken down in a great epidemic, the direct mortality of the disease must be said to be slight. The prognosis, however, is serious when the patient attacked is already suffering from a chronic disease like diabetes, nephritis, valvular affection, or phthisis, even though the influenzal attack seems to be but a mild one. Pneumonia is the great factor in the mortality, and the outlook in a given case must be based upon the physical signs in the lung, and especially upon the condition of the heart. It is a remarkable feature in influenzal pneumonia how frequently recovery follows in aged subjects, who would naturally be expected to succumb to a true croupous attack; though upon the whole the prognosis must be regarded as grave, even in the majority of such subjects, owing to the indefinite and often prolonged course of the disease. The prognosis should always be guarded in those patients suffering from relapses or complications who have not taken to bed during the period of onset, as a fatal issue often follows trifling exposure.

Treatment.—The obvious prophylactic treatment consists in the avoidance of infection, when this is possible.

Rest in bed as soon as the first symptoms of the disease become apparent, is of the greatest importance in every case, mild or severe. Of drugs, none give such marked relief as antipyrin ; 5 grs. in solution in chloroform water every three or four hours soon relieve the headache, backache, and joint-pains, and it is rarely necessary to continue the drug beyond twenty-four or forty-eight hours. The complications are to be treated on recognised general principles, the pneumonia requiring usually a free administration of alcoholic stimulants, and cardiac failure demanding strychnine hypodermically. Quinine and salicylates are praised ; the former drug in severe pneumonic cases, when given with full doses of ammonia, affords the best routine treatment after the relief of the initial fever by antipyrin. The nutrition of the patient must be carefully watched. The greatest care must be exercised in permitting the convalescent to move out of doors,

pulmonary symptoms being in the great bulk of the cases caused by exposure before convalescence has been thoroughly established.

The early free incision of the thoracic wall where empyema occurs gives excellent results, even in the most unpromising cases.

INSOMNIA

Sleeplessness cannot be regarded as a disease ; it is but a symptom of many diseased conditions, and when not the direct or immediate result of severe pain or acute discomfort it may be usually considered, as held by Professor Bradbury, to be due to an altered metabolism of the cerebral cells, dependent upon exhaustion and diminished influx of stimuli. The disturbance of metabolism is probably the result of poisoning by excrementitious fatigue-products.

Vascular disturbance, by which from loss of tone the vessels supplying the cerebral tissue remain dilated, thus causing a hyperæmia of the brain, is a potent factor in many cases. This dilatation may be the result of a severe general anæmia or of toxic influences, or it may arise from some cause which shuts off the blood-supply from a distant region, thereby permitting an undue influx of blood to the brain, the normal condition of which, during sleep, is supposed to be that of anæmia. This is obvious in those cases where coldness of the feet keeps the patient awake till artificial warmth is applied to the extremities.

Various attempts at a classification of the different types of insomnia have been made, all of which have been unsuccessful or unsatisfactory, since the same causes may be operative in apparently widely different forms of sleeplessness. The division into 'primary' and 'secondary' is, however, convenient, since under the latter term can be included all those cases which depend upon pain or physical suffering, cough, frequent micturition, pruritus, or any peripheral irritation or external stimuli.

The so-called 'primary' form of insomnia will include the cases of *psychical* origin, which are the result of mental excitement, worry, grief, shock, &c. ; to this type belongs the insomnia so frequently met with in neurasthenia, hypochondriasis, melancholia, and other forms of insanity. The sleeplessness caused by mental over-work or study, especially when this is protracted up to the hour of retiring to bed, belongs to this group. The *toxic* form, in which a poison introduced from outside the body is the cause of the banishment of sleep, is a very common one ; it includes those examples of insomnia dependent upon the excessive use of tobacco, alcohol, tea, coffee, &c. Allied closely to it is the sleeplessness produced by poisons generated within the organism by

such germs as cause the infectious fevers, pneumonia, influenza, &c., or by the deleterious products manufactured in the gouty, rheumatic, or dyspeptic processes.

A form of sleeplessness, which in the absence of a better word might be termed 'habit-insomnia,' is frequently met with, in which insomnia has been induced by some of the causes belonging to the category above mentioned, and after the cause has been removed the patient finds that he has acquired the habit of lying awake. This habit-form of insomnia is observed in persons who from some unavoidable cause have been compelled to change their hour of retiring to rest; sleeplessness follows, and finally becomes chronic, even though the original hour be returned to. To this form of insomnia are allied those cases where a person accustomed to sleep soundly in the vicinity of loud noises or the hum of continuous machinery suffers from sleeplessness when deep silence is maintained.

Symptoms.—The sleep may be disturbed, broken, fitful or restless, or interfered with by unpleasant dreams or terrifying nightmares, but usually the term 'insomnia' is reserved for those severe degrees of sleeplessness in which no sleep is present for considerable periods, or in which the patient lies awake for hours after retiring to rest, and only falls asleep towards morning. Another most serious form exists in which the patient sleeps immediately after retiring to rest, but awakes after an unconscious period of minutes' or hours' duration, with his brain abnormally active and wide awake—a condition which may continue till his getting-up hour arrives.

Partial or incomplete insomnia is much more common than the complete form, and though patients sleep more than they themselves believe, still, examples of the condition are not infrequently observed in which the victim of complete insomnia may continue awake for several days and nights in continuous succession. The writer knew a late distinguished scientist who habitually suffered from insomnia in a most aggravated form, and in one attack no moment of sleep was obtained for fourteen consecutive days and nights. In such rare cases the chronic condition may well be regarded as a 'disease,' since no symptom of any primary malady may be observable.

Treatment.—This will be found detailed in the last edition of the author's 'Dictionary of Treatment,' upon pages 459 to 472; only the general principles can be here mentioned. The first duty of the physician is to find out the cause of the insomnia by a minute investigation of the patient's physica condition and of his habits. In the great majority of cases evidence of the transgression of some important health-law will be forthcoming, especially as regards the amount of brain output or worry. If the cause is removable, little

drugging is needed save the administration of a pure hypnotic like sulphonal, paraldehyde, trional, or full dosage by bromides, for a short period, to break the habit of lying awake. All such drugs are, however, valueless in the presence of pain, which first must be relieved by morphia or other narcotic.

When sleeplessness is the result of such conditions as delirium tremens, fever, heart-disease, melancholia, mania, gout, Bright's disease, &c., the obvious indication is to treat the primary cause when possible, and relieve the insomnia from time to time by narcotics or hypnotics when it threatens to exhaust the patient's strength. The insomnia of acute pneumonia and bronchitis must not be relieved by morphia or opium. Many deaths, it is to be feared, are caused by narcotics in these and other allied conditions, and chloral is especially dangerous in the sleeplessness of heart-disease. Paraldehyde is always safe. The special indications for douches, massage, electricity, hot baths, &c., and the relative values of the members of the ever-flowing stream of new hypnotics, will be found discussed at considerable length in the article already referred to.

INTESTINAL CANCER

The disease pursues the same course as it does in other parts of the body, if we omit its tendency to cause obstruction. The diagnosis, prognosis, and treatment of this affection will be dealt with in the succeeding article on intestinal obstruction.

Etiology and Pathology.—The large intestine, including the rectum, is about ten times more frequently involved than the small intestine. In the great majority of the cases the disease is primary, and of the cylindrical-celled epitheliomatous type, the round-celled form being rare, though both may become colloidal. Sarcoma of the intestine is so rare that it may, in all cases of doubtful diagnosis, be left out of question. Intestinal cancer may appear as a diffuse or nodular neoplasm, which tends to involve the peritoneum, lymphatic glands, liver, and other organs; but its most usual form of extension in the bowel is an invasion of the intestinal coats in an annular manner, which produces stricture, leading to blocking. Ulceration is prone to occur above or at the site of the stricture.

If pyloric cancer be included in the list of gastric diseases, then duodenal cancer must be said to be comparatively rare; but it is a remarkable fact that the first ten inches of the intestine are invaded slightly more frequently than the succeeding eight feet of jejunum, and almost as frequently as the thirteen or fourteen feet of ileum, whilst the cæcum is the seat of malignant disease about as frequently as all the portions of the small intestine taken collectively.

Symptoms.—Intestinal cancer will betray its presence by the characteristic cachexia of malignant disease; by its tendency to cause intestinal obstruction; and often by the presence of a tumour palpable through the abdominal walls.

Duodenal cancer presents itself under two well-marked clinical types. Where the new-growth is situated in the first part of the tube above the entrance to the bile-duct, the symptoms are in the main those of pyloric cancer, with a movable tumour, dilatation of the stomach, pain, vomiting, cachexia, &c. When the obstruction is below the biliary opening, both bile and pancreatic secretion are discharged into the upper dilated portion of the tube, from which they regurgitate into the stomach, and always appear in the ejecta. Though bile is absent or deficient in the stools, there is no jaundice, by which sign the disease is differentiated from cancer of the head of the pancreas, though the tumour is fixed or immovable. However, if the malignant deposit block the end of the common bile-duct there will be signs of jaundice, which becomes intense and persistent (see page 430).

The diagnosis of cancer of the small or large intestine is often obscure or impossible till the presence of a definite tumour or obstructive symptoms supervene. The age, history, wasting, and the appearance of the typical cachexia should excite a strong suspicion, which can be converted into a certainty only by an exploratory incision.

Treatment.—This will be dealt with under 'Intestinal Obstruction.' Early removal of the site of the strictured portion of the bowel will prevent obstruction and prolong life very considerably; when this cannot be accomplished, the establishment of an anastomosis may give great relief. Gastro-enterostomy in duodenal cancer is often followed by most satisfactory results. In the case of the colon short-circuiting and colotomy, when colectomy is impossible, are indicated.

INTESTINAL OBSTRUCTION

Acute arrest of the passage of the intestinal contents towards the anus is produced by several distinct causes: (1) Intussusception; (2) strangulation; (3) volvulus; (4) strictures; (5) tumours within or without the bowel; (6) accumulation of fæces or foreign bodies; (7) paralysis of the bowel or so-called paralytic ileus.

Chronic intestinal obstruction may be the outcome of the acute form in rare cases. Usually it is the result of the following causes: (1) Stricture of the small or great intestine, simple or malignant; (2) tumours within or without the bowel; (3) adhesions gluing together coils of the small intestines, as

in tubercular peritonitis ; (4) foreign bodies or fæcal accumulations ; (5) chronic intussusception.

Whilst the division of intestinal obstruction into 'acute' and 'chronic' is convenient, it must not be forgotten that an important group of cases exist in which acute symptoms come on with great intensity and abruptness during the course of the chronic malady.

The relative frequency of the above causes is stated most diversely by different authorities, according as the statistics are based upon *post-mortem* records, operative cases, or tables of the cases generally met with in practice. Thus, if the latter method of summarising the causes be accepted, accumulations of fæces will be found to be a common source ; whilst if the acute fatal groups be analysed, the most frequent cause of obstruction, as is evident from the figures of Sir Frederick Treves, will be found to be intussusception, which is the cause of death in slightly more than one-third of all the fatal cases, strangulation following as the origin in one-fourth of the total, and stricture in one-sixth to one-seventh, a similar proportion being the result of foreign bodies and tumours within or without the bowel ; whilst volvulus only accounts for one fatal case in every twenty, and fæcal accumulation is the cause in about one case in every seventeen fatal cases.

ACUTE OBSTRUCTION

Intussusception.—In this condition a portion of intestine, having its calibre temporarily diminished, slips into the wider portion below it, as the finger of a glove is often seen to become invaginated in rapidly ungloving the hand. In this way three parallel layers of bowel are involved in the tumour formed by the invagination. Looked at in a longitudinal section from within outwards, the mucous membrane of the upper portion of the intestine will be found in direct line with the mucous lining of the tube on the stomach side of the intussusception. This innermost layer is known as the 'entering' layer. Its serous surface is in direct contact with the serous coat of the middle layer, which is known as the 'returning' one. The outer layer of bowel is called the sheath, or the 'receiving' layer, or the intussuscipiens, since it receives the entering and returning layers, which together form the invaginated portion or intussusceptum. The mucous surface of the sheath is in direct contact with the mucous surface of the middle or returning layer. If the edge or face of a transverse section of the flattened-out invagination be examined from without inwards, the following strata of tissue will be met with in the order mentioned—viz. : (1) Serous membrane ; (2) intestinal wall ; (3) mucous membrane ; (4) mucous membrane ; (5) intestinal wall ; (6) serous membrane ; (7) layer of mesentery ; (8) serous membrane ;

(9) intestinal wall: (10) mucous membrane—bounding the lumen of the tube, on the other side of which will be found, of course, the same strata in inverse order from within outwards. The 'apex' of the intussusception is situated at the inferior end of the entering layer, and the 'neck' at the upper part where the returning layer joins the sheath.

In some cases, the layers having become more or less adherent, the entire tumour may slip into the cylinder of bowel below it, and become again invaginated, forming a double intussusception; and even a triple form occurs sometimes.

Any part of the small or large intestine may be the seat of the invagination, the most common form being that in which the ileum and cæcum pass downwards, with the ileo-cæcal valve in front, into the colon. In one case seen by the writer, a papilloma of the jejunum caused an acute intussusception by invaginating many yards of small intestine, the papillomatous growth which formed the apex of the tumour presenting at the anus, where it was grasped by the sphincter.

Intussusceptions occur immediately before death in various diseases, most commonly in children dying from tubercular meningitis. They are always limited, and draw out on the slightest traction applied to either end. They cause no symptoms, are often multiple, and are of no clinical importance. They are probably the result of inverted peristalsis, as they usually occur from below upwards, whilst in the serious form the upper portion of intestine almost always slips into the lower.

The mesentery, as already stated, is found between the inner and middle layers of the intussusceptum; it drags upon the tumour, so that this becomes curved, and the traction further tends to cause a narrowing of the orifice of the innermost cylinder, which may be converted into a mere slit. This, with the congestion and swelling which ensues, finally blocks the bowel, and, as the congestion increases, hæmorrhages into the walls of the intussusceptum follow, with great œdema, and finally the low form of inflammation thus induced ends in gangrene of the portion of gut invaginated. This may become detached and, passing down the bowel, may be expelled with the fæces.

The swelling may be so great in a few hours as to render reduction of the invagination difficult or impossible, and the serous coats may become united by rapidly formed adhesions which, after a few days, may make the surgical reduction an impossibility without laceration or rupture.

The condition is met with chiefly in young children, where it is supposed to be caused by vigorous and irregular peristalsis, often associated with purgation, the ileum being prolapsed through the ileo-cæcal valve as the rectum is prolapsed so frequently in children through the anal sphincter.

STRANGULATION.—Here the bowel becomes nipped by a loop or knuckle of it slipping under some rigid unyielding band, or by entering some aperture or slit in the mesentery or omentum, or by its becoming involved in an internal hernia at the diaphragm, foramen of Winslow, or elsewhere. The bands which cause the strangulation are endless in their variety. Some are of congenital origin, as the diverticulum of Meckel, which may remain as a fibrous cord containing the remains of the vitelline duct, which stretches from the ileum near the ileo-cæcal valve to the umbilicus. Allied to these are the bands which are formed by the tip of the appendix, or a Fallopian tube becoming adherent to some structure in its vicinity. The usual source of the bands or cords is some previous peritonitis, which has resulted in adhesions; the adhesion, whilst remaining fixed to some structures at each end, becomes gradually lengthened by the normal visceral movements, so that a band or cord is formed free in every respect, save at its attached extremities; under the bridge so produced a loop of bowel slips and becomes insnared. A prolific source of such bands is old appendicular disease, or the local peritonitis following previous abdominal sections, or the inflammatory action produced by tubercular deposits.

In the group of strangulations causing acute abdominal obstruction must also be included those kinkings of coils of intestine which are matted together by inflammatory products, fixing the bowel in an acutely bent position, which then, like a bent rubber-tube, becomes completely obstructed. A band, ribbon, or cord, attached to a fixed point at one end and to a movable piece of intestine at the other, may, when any traction is made upon it, cause a kink or acute bend, which for the time obliterates the lumen of the gut. The occasional connection of these bands with old hernial sacs must be remembered, one end being commonly attached to the omentum and the other to some place in the neighbourhood of the old hernia.

The exact manner in which the intestine becomes strangulated is as variable as are the varieties of the bands and cords. The pathological changes at the point of constriction, and in the intestine above it, are in the main similar to those observed in strangulated femoral and inguinal hernias. The inflammatory action set up in the coats of the intestine above the constriction causes such infiltration of the damaged tissues with the virulent bacilli always present in the bowel, that septic poisoning develops rapidly, from which the patient may succumb even before signs of perforation or general septic peritonitis have time to appear.

VOLVULUS, or TORSIONS, are comparatively rare. The most frequent site is the sigmoid flexure, the coil of bowel being twisted round an axis formed by its own mesentery, or

in some cases it may be obstructed by a coil of small intestine twined around it. The former condition is much more frequent. The circulation is blocked by the torsion of the mesentery, and the lumen of the gut is occluded by one end of the coil pressing upon the other end, as in these cases the meso-colon is always long, and both ends of the sigmoid loop are in close juxtaposition. This form of obstruction occurs only in elderly adults, much more frequently in males than in females, and is probably the result of a chronic constipation, which causes the sigmoid to become distended and displaced towards the pelvis. The loop of affected bowel rapidly distends with gas, and may fill the entire abdomen, pushing up the diaphragm and displacing the viscera. The intestinal walls become rapidly chocolate-coloured, gangrenous patches appear, and a grave septic peritonitis speedily develops. This form is most acute in its onset, and runs a very rapid course. Volvulus of the cæcum and small intestine also occurs, caused usually by a twisting of the bowel round its own mesenteric axis, though occasionally a coil of small intestine twists or entwines itself round another coil or long loop, which serves as an axis for it.

Strictures.—These are much more frequently the cause of chronic obstruction than the origin of acute blocking; though often the onset may seem sudden, an investigation of the history of the case will reveal that the attack is an acute one on the top of a chronic.

Congenital malformations about the rectum are often seen, and atresia of the pylorus is much more common than is generally supposed. The imperforate condition of the anus or rectum leads to urgent symptoms of abdominal obstruction soon after birth.

Strictures may be caused by malignant disease, usually of the epithelial type, in which an annular ring surrounds the lumen of the gut and blocks it. In other cases the stricture is the result of the gradual contraction of the cicatricial tissue, which has been produced during the healing of typhoid, tubercular or syphilitic ulcerations, or old injuries caused by hernia or foreign bodies. The bowel-stricture causes total obstruction in many cases where *post-mortem* examination reveals that the lumen of the tube had not been closed. This result would appear to be somewhat like that seen in urethral stricture, where complete retention supervenes on a moderate narrowing of the passage.

The bowel above the stricture becomes dilated, sometimes ulceration takes place in it, and perforation is liable to occur. Thus, in one case under the writer's care, a child with suspected stricture of the small intestine was placed upon a diet restricted to fluids; she managed to procure an orange, and after greedily eating it was seized with violent symptoms

of peritonitis and abdominal obstruction. The *post-mortem* revealed an aperture in the dilated part of the bowel above a cicatricial stricture, through which a sharp-pointed pip of the orange had passed.

Volvulus and kinking may occur in the bowel above the narrowed part, and they may set up acute obstruction in a case where stricture has never been suspected.

Tumours.—Polypi are not uncommon, and may be multiple, and, like the rare sarcomata of the spindle-cell type, may occasionally produce symptoms of acute obstruction like those following stricture. They are liable to lead to intussusception, as in the case mentioned under 'Intussusception.' Tumours in the abdomen outside the bowel, especially when originating in the pelvis, may by their pressure block the lumen of the intestine. Ovarian growths are liable to act in this way, and large uterine fibromata not rarely cause complete blocking of the sigmoid or rectum.

Accumulation of Fæces and Foreign Bodies in the Intestine.—The colon becomes distended with fæces in chronic constipation and in gross feeders who do not take time to masticate their food, but bolt it in large pieces. This chronic condition belongs to the category of chronic obstruction in many cases, but often the cæcum or sigmoid may be permanently loaded with fæces, whilst the patient has a motion every day, only a mere fraction of the mass being expelled. Much oftener the scybalous masses may be tunnelled with a central hole, through which loose motions are passed. The intestinal wall is the seat of a low form of inflammation, and often ulcers occur. At any time the symptoms of acute obstruction, followed by a rapid septic or perforative peritonitis, may supervene in a case where the primary chronic condition had never been suspected.

In other cases acute obstruction may supervene after a child has gorged itself with nuts, gooseberries, &c. The writer knew of one fatal case which followed eating a large amount of jujubes, and another instance in which a physician had swallowed a quantity of preserved sugared fruit.

Though these latter examples may be regarded as illustrations of foreign bodies, this term should be restricted to substances which, from their nature, are indigestible or insoluble, as artificial teeth with their metal fittings, wool, hair, buttons, pins, needles, rings, &c., cherry- and peach-stones, &c. The usual seat of lodgment is the cæcum; less frequently the duodenum and rectum. Gall-stones which pass through the common duct into the intestine are extremely unlikely to cause intestinal obstruction. Large calculi which have ulcerated their way directly into the duodenum, small intestine, or colon from the gall-bladder very frequently have caused acute intestinal obstruction. Though large calculi

are often passed per rectum without difficulty, it is a well-recognised fact that acute and fatal intestinal obstruction is sometimes produced by comparatively small stones—a phenomenon of which no adequate explanation is forthcoming. The usual site of the obstruction in such cases is the lower portion of the small intestine above the ileo-cæcal valve.

Enteroliths, or rounded concretions formed of lime-salts, matted hair, the awns of cereals, or masses of medicinal substances, as magnesia and salol, have been known to cause acute obstruction, acting like gall-stones.

Paralysis of any part of the intestinal tube below the stomach may cause acute obstruction. Such a result is not very rare after the successful release of a strangulated femoral or inguinal hernia, or after an abdominal section. The condition may be more general after severe injuries of various kinds, which induce a paralytic state of a large portion of the intestines, followed by great distension of the tube; this leads to active mechanical obstruction, according to Morison, by producing acute bendings at every few feet of the bowel. To such a condition the name Paralytic Ileus is applied.

Symptoms of Acute Obstruction.— These come on with abruptness, in the majority of cases the onset being very sudden. Pain is the first warning of anything being wrong; nausea follows in a few minutes, which, after a variably short time, ends in vomiting, and more or less collapse is present early. Tenderness may be elicited at an early stage over some localised area, but it soon becomes general after the abdomen has become tympanitic and the vomiting stercoraceous. There is usually complete constipation; seldom, if ever, any flatus passes from the anus. There is no fever at first, and indeed all through the attack the temperature may remain subnormal, even in the face of a fatal septic peritonitis. Thirst adds greatly to the suffering; the tongue is foul, the pulse from the first is rapid, but soon becomes weak, and in the late stages thready and uncountable. The amount of urine is always markedly diminished, and death usually takes place from septic peritonitis before the end of a week.

Pain, which is usually the earliest symptom, may be unmistakably localised for the first few hours of the attack. Generally when the case falls under the physician's observation the pain is either referred to somewhere near the umbilicus, or it is vaguely localised or felt over the entire abdomen. In some cases this diffuse pain may become localised after a short time. When there is marked *tenderness* confined to a definite area, as that of the cæcum or sigmoid, and when the tenderness and pain correspond in their distribution, the combination is of much value in localising the site of the lesion; but pain alone is apt to mislead if depended upon, especially if the pain is relieved, as it usually is, by pressure. The

nature of the pain in acute obstruction is of great importance. From the very first it may obviously be of the intermittent type, which is caused by exalted and ineffectual peristalsis. Intelligent patients will often volunteer the statement that it resembles the severe colicky pain which accompanies or precedes the action of a strong griping purgative. Sir F. Treves lays stress upon the rule that when the pain is constant, though liable to periodic exacerbations, the obstruction may be regarded as complete; and when it is distinctly intermittent—*i.e.* when there are short intervals of total absence of pain—the obstruction may be regarded as only partial. These features in the nature of the pain, the same authority states, will often distinguish a severe strangulation from a stricture.

The sudden disappearance of pain at a late stage is a very grave symptom, which may indicate perforation into a neighbouring coil of bowel; whilst its gradual fading away in the presence of a deepening of the constitutional symptoms may be taken as indicative of the onset of a rapidly fatal peritonitis.

Poulticing gives some relief to the suffering, but pain is always aggravated by everything which increases the peristalsis, as rough handling, purgatives, and irritating enemata.

Vomiting may be said to be never absent in acute intestinal obstruction; its severity is, generally speaking, proportionate to the length of the intestinal tube below the obstruction. Thus, in blocking of the upper part of the small intestine the vomiting is very copious, frequent, and always bile-stained, and the fluid is ejected with considerable force, as in sea-sickness. When the lower part of the small intestine is blocked, the ejecta becomes tainted with a suspicion of fæcal odour, which, after some hours, is quite unmistakable. A distinctly foul fæcal odour is common without any blocking below the ileo-cæcal valve, and the typical stercoraceous vomit is generally obvious before the fifth day in cases where the obstruction is confined to the small intestine below the lower two-thirds of the ileum. In blocking of the colon the fæcal contents of the great intestine may regurgitate through the valve, and in this case the stercoraceous vomiting usually does not occur so early as when the lower part of the ileum is involved. and it is never very copious.

Stercoraceous or fæcal vomiting, though highly characteristic of intestinal obstruction, is nevertheless not pathognomonic, as it is occasionally noticed in malignant disease which has ulcerated and formed a fistula between the stomach or duodenum and the colon.

Urinary diminution is usually in proportion to the copiousness of the vomiting; as a rule, the site of the obstruction does

not markedly influence it. Treves regards it as a collapse-symptom caused by the severity of the obstruction. Though the thirst is burning and insatiable, the fluids are so rapidly ejected that little is absorbed.

Flatus is abundant, and may be belched up through the mouth, or it may cause great distension of the abdomen with borborygmi, but it is never passed through the anus in complete obstruction, except in some cases of intussusception, during violent straining.

Constipation is generally absolute from the onset, except in intussusception. Occasionally a motion may occur after the seizure; but as a rule, unless enemata are administered, the colon retains the fæces present when the pain first set in, though the strangulation may be situated high up in the small intestine. As in some cases of hernia, the constipation may be complete without the entire lumen of the bowel being involved, and it may be absolute in paralytic ileus.

In some very rare cases, diarrhœa may be present all through the attack, owing to an inflammatory condition of the mucous surface below the obstruction, and in intussusception and strictures just above the rectum, tenesmus and the passage of bloody mucus may simulate diarrhœa. Some diarrhœa may be present in an acute obstruction supervening upon a chronic condition in which the entire colon is blocked with scybalæ, which have been tunnelled.

Abdominal distension sets in some hours after the onset. During the interval the abdominal walls are retracted. The degree of distension varies greatly; it comes on rapidly and becomes extreme in volvulus of the sigmoid coil, and as a rule it is more marked when the colon is the seat of the obstruction than it is in blocking of the small intestines.

In chronic cases of obstruction the hypertrophied and distended coils of bowel may be seen and felt through the distended abdominal wall in their rhythmical attempts to drive on their contents through the narrowed or blocked portion. In acute obstruction high up in the small intestine there may be very little distension of the abdomen.

Constitutional symptoms may betray the gravity of the situation from the moment that pain is first felt, as seen in those cases setting in with signs of shock or collapse, pallor of the face, pinching of the features, with sunken eyeballs and cold, clammy perspirations. The pulse affords a reliable criterion of the seriousness of the condition: it is quick from the start, but rapidly weakens and becomes thready. Though the temperature is usually sub-normal throughout, the feeling of intense thirst is incessant. The tongue in the early stages is covered with a moist fur; later on it becomes red, dry, and finally brown. Death usually occurs before the end of a week, but in severe cases of complete obstruction it may occur

as early as the third day. The patient sinks into the condition which is usually met with in septic peritonitis, and dies from pure exhaustion caused by constant vomiting, hastened by the absorption of toxins produced by the bacilli which invade the intestinal walls and the peritoneal cavity. Coma before death is rare and delirium is uncommon, and to the last the temperature may remain sub-normal. An evacuation of the bowel is not uncommon before death.

Diagnosis.—The classic symptoms of pain, shock, vomiting, absolute constipation, &c., coming on suddenly, as just described, in the great majority of cases will settle the diagnosis of acute intestinal obstruction.

Peritonitis resulting from appendicular disease is perhaps more frequently confused with acute obstruction than is any other affection. The first point in the diagnosis is a careful investigation into the patient's history, which may elicit the fact that several previous attacks of a similar nature had occurred—a history only to be obtained in recurring appendicitis. In the peritonitis of appendicitis the temperature is always well above normal from the first day, though it may sink at a later stage. The pain is always less acute, and is more continuous; seldom does it display a marked rhythmical quality, since it is the pain caused by inflammatory action and not that produced by ineffectual peristalsis, and it is seldom described by the patient as colicky.

Symptoms of shock or collapse are absent, save at a late stage when an abscess has burst. Constipation is very seldom absolute, and flatus may be expelled by the anus. Vomiting is never stercoraceous ; often vomiting and nausea are entirely absent. The abdomen is not distended ; its walls are usually rigid and very tender, and no coils of distended bowel mark any part of the surface. The location of the abdominal signs in the appendix region is a matter of considerable importance, though mistakes are liable to be made in any diagnosis arrived at chiefly on a regional basis. The presence of the perityphlitic abscess at a later stage is characteristic. Though appendicitis is often mistaken for acute intestinal obstruction, the latter is seldom mistaken for appendicitis. When perforation of the appendix suddenly occurs in latent cases, the general septic peritonitis resembles that seen in the last stages of obstruction.

Perforation of a gastric or duodenal ulcer produces symptoms of a rapid general peritonitis, which can only be mistaken for a case of obstruction when first seen in its last stages. The history usually settles the diagnosis. The same remark applies to the peritonitis caused by *ectopic gestation.*

Cholera has been mistaken for a severe acute complete strangulation of the upper half of the small intestine ; the chief danger of error lies in the interpretation of the obvious

signs of shock and collapse which may usher in both diseases. The pain is never so severe in cholera at the onset as it is in obstruction, and the appearance of the rice-water stools is unlike anything met with in those rare cases of obstruction setting in with diarrhœa.

Acute dysentery and *acute enteritis* have been often confused with intestinal obstruction caused by intussusception, but the clinical features of these diseases are usually quite distinct. When enteritis follows abdominal operations the paralysis of the bowel, as in paralytic ileus, may cause it to rapidly distend with gas, so that a number of acute bendings occur which may produce an acute mechanical obstruction.

Hepatic and *renal colic* are only liable to be mistaken in the first onset when collapse-symptoms may be unusually severe; but the profuse vomiting which occasionally follows an impacted gall-stone may, in nervous patients, closely simulate an intestinal obstruction high up. The peculiar rhythm of the pain and the appearance of jaundice in twenty-four hours, coupled with a previous history of similar attacks, are usually distinctive. The changes in the urine, retraction and pain in the testicle, and other local symptoms differentiate renal colic.

Hernia produces symptoms identical with those of acute intestinal obstruction, and every case of severe abdominal pain and vomiting demands in the first instance a careful routine examination of the femoral, inguinal, and umbilical regions. The frequent association of internal strangulation with bands attached to old hernial sacs has been already emphasised.

The diagnosis of acute intestinal obstruction having been established, the next points requiring consideration are, What is the nature of the obstruction ? and secondly, Where is it situated ?

Intussusception presents the following diagnostic features, many of which are quite characteristic. It is a disease of early youth or childhood, half of all the cases occurring before ten years, and a quarter before the end of the first year. The symptoms are those already described as common to all forms of acute obstruction—viz. pain and vomiting, with suddenness of onset. The continuous pain in this variety of obstruction is at first often as severe as in strangulation, but the colicky feature develops markedly, with long intervals at first in which pain may be totally absent. In some instances the pain bears a miniature resemblance to labour-pains. The vomiting is seldom very copious or severe, and is absent in rare cases. It may become stercoraceous, and a peculiar feature in it is that, unlike the vomiting seen in strangulation, often relief is afforded for a considerable time after the contents of the stomach are ejected ; but this symptom is also seen in

volvulus. Constipation is sometimes present throughout; but the most distinguishing feature in intussusception is a diarrhœa in which small loose motions are passed with much bloody mucus and great rectal tenesmus. The writer has seen this latter symptom in one adult case to be so intense as to cause screaming like that met with in some severe cases of gall-stone colic. Flatus is constantly passed; the abdomen is seldom distended; in a very considerable percentage of cases a sausage-shaped tumour may be seen or felt, usually about the region of the descending or transverse colon, and occasionally about the ileo-cæcal valve, and the pain usually becomes localised at the site of the tumour. The tumour may be felt by the finger passed into the rectum; it is not markedly movable, and it may be noticed to get firmer during an attack of pain, and occasionally it may be only palpable during the paroxysms of pain. Sometimes its slow progress from the valve towards the sigmoid has been observed, and in a considerable number of cases it is found at the anus before the attack has lasted a week.

The journey of an ileo-cæcal invagination along the colon to the rectum in an acute case in an infant may only occupy twenty-four hours.

The constitutional symptoms are not severe, but in some acute cases there may be a considerable degree of shock.

Strangulation, which is the cause of about as many cases as is intussusception, affords the most typically acute form of obstruction. The symptoms are those which have been described as common to all the varieties; and often the only means of differentiating the obstruction depending upon bands, cords, &c., and internal hernia from the other forms is by the suddenness of the onset, the rapidity of the progress, and the severity of the symptoms, especially of the stercoraceous vomiting and diminution of the urine, and the absolute degree of the constipation; there is usually slight distension. A history of previous general peritonitis or of abdominal section, or the presence of old hernial sacs, may suggest a clue. The history of an old appendicitis is suggestive of the cause being an internal strangulation, and old Fallopian-tube mischief points in the same direction. A large diaphragmatic hernia may give clear thoracic signs. The condition is much more frequently met with in strong muscular males before twenty.

Volvulus may be very rapid in its progress and sudden in its onset; usually the distress is referred to the left side in the sigmoid region. Though the constitutional symptoms may be very severe the vomiting is usually not so marked. It is seldom stercoraceous, and is often followed by some relief. A characteristic feature is the rapidity with which abdominal distension supervenes, and this reaches a degree which is hardly

ever seen in other forms. Generally nothing can be felt on rectal examination, but there is often marked tenesmus, though the constipation is complete and tenderness over the distended sigmoid is usually present.

This form is nearly always met with in elderly males who are the subjects of chronic obstinate constipation ; it is very rare in women, and in males before forty-five.

Stricture and *tumours*, when they cause acute obstruction, may often be differentiated from the foregoing varieties by a careful scrutiny of the patient's previous history. In the majority of cases there will be found evidence of a minor degree of chronic obstruction, which has been often mistaken for constipation, and the coils of hypertrophied bowel may be visible above the obstruction when first seen. Malignant strictures are associated with the cancerous cachexia, and the history may reveal evidence of tubercular, typhoid, or syphilitic ulcerations in other cases. As a rule, this group is best included in that of chronic obstruction, but it is here referred to to emphasise the fact that not rarely the physician is first called upon to deal with an apparently acute obstruction whose previous symptoms have been overlooked.

Gall-stones which have escaped through fistulous communications between the gall-bladder and intestine may produce sometimes symptoms quite as acute as those met with in the worst varieties of strangulation. The diagnosis is obscure, but a history of old gall-bladder trouble or of hepatic colic, if discovered, is of great value. The vomiting is exceedingly copious when the stone blocks the upper part of the small intestine. The presence of a gall-stone obstruction may be suspected when the symptoms suddenly disappear, with relief of all the constitutional disturbance, and the concretion may be found afterwards in the motions. Another point of value in the diagnosis is the frequency with which women are affected; the subjects are usually obese and generally above fifty. In the majority of gall-stone impactions, however, the obstruction is seldom complete.

Fæcal accumulations and *foreign bodies* as a rule produce the same clinical features in their acute symptoms as do gall-stones and strictures—*i.e.* they illustrate the type of chronic incomplete obstruction which is liable sometimes suddenly to assume acute gravity, especially when it has remained latent for a considerable period. Moreover, a small mass of fæces, a foreign body, or some indigestible food may be caught in a stricture of slight degree, and acute symptoms supervene suddenly. In other cases a slight perforation of the dilated bowel above the stenosed portion may cause a local or general peritonitis of sudden onset and gravity, or kinking may occur at the narrow part, converting suddenly a chronic into an acute condition.

The second point in the diagnosis of acute obstruction may be even more difficult than the discovery of the cause. It is often impossible to be certain where the obstruction is located. Pain, as already stated, gives uncertain information; tenderness is more reliable, and when both coincide, as in intussusception, with the evidence of a tumour, the difficulty may be said to be solved.

When the small intestine is the site of the obstruction, as already mentioned incidentally in the general description, the onset is more abrupt, the vomiting more severe, and the pain more acute than when the colon is blocked, the exception being sigmoid volvulus.

When the upper part of the small intestine is blocked there is usually little distension ; when the lower portion is occluded the abdomen is rounded like that seen in ovarian tumours, and after the middle of pregnancy. Blocking of the colon always causes much distension; and this at first is noticeable in the flanks, which bulge enormously, leaving the central region flat. In volvulus of the sigmoid, as already stated, the distension, starting on the left side, rapidly assumes colossal proportions, and becomes general. As a rule, in chronic obstruction the localisation is much easier, since the distended and hypertrophied coils of bowel may often be mapped out on the proximal side of the block.

No help, as a rule, is obtainable from measuring the amount of water which can be pumped into the colon, and the long tube should never be employed. Rectal examination gives little help. The rectum is usually ballooned, especially in chronic cases ; but if tightly distended coils of bowel can be clearly appreciated by the finger-tip, the obstruction is likely to be in the small intestine.

Much has been written about the differential diagnosis of the cause and of the localisation of the obstruction in the acute forms, but the question is not one of vital importance. Once an acute obstruction is diagnosed, the duty of the physician is without delay to hand the case over to the surgeon for operative treatment. To dally from hour to hour or from day to day, waiting for a diagnosis of the particular cause, is in many cases little less than criminal: the patient's chances of recovery depend largely upon the promptness with which surgical procedures are undertaken. Very often the surgeon finds a totally different state of affairs in the abdominal cavity from what the symptoms had led him to expect ; nevertheless the physician should leave no stone unturned to discover the cause of the obstruction, but he should not jeopardise the patient's chances of recovery by postponing the operation for this purpose.

Prognosis.—This is always most grave. All the strangulation cases will probably die if not relieved surgically before

the end of a week; some succumb inside twenty-four hours. Intussusception in infants may prove fatal in a few hours; but the duration of this form is usually somewhat longer than in strangulation, and a considerable percentage of them may linger on for weeks, and not a few recover by the invaginated portion being expelled per rectum, as in the chronic form of the disease. Volvulus of the sigmoid is quite as fatal, and generally almost as speedily fatal, as strangulation by bands, and the same is true of some gall-stone cases. The cause of death in the majority of cases is septic peritonitis; often it is hastened by perforation, following gangrene or ulceration of the bowel above or about the site of obstruction: perforation usually resulting above the strictured spot, and gangrene affecting the entire strangulated loop or the invaginated portion of the bowel below the constriction.

Once fæcal vomiting supervenes, death usually follows within sixty or seventy-two hours. The prognosis then for all practical purposes may be regarded as necessarily fatal in every form of acute obstruction without surgical aid. The escape of one in seven cases of acute intussusception by spontaneous recovery through the discharge of the gangrenous bowel by the rectum should not influence the decision for early operation. The sooner the physician realises that every case of acute obstruction of the bowel calls for as speedy surgical aid as does a strangulated femoral hernia, the better for his patient.

Treatment.—*In strangulation cases.*—Unquestionably some of the most unpromising cases even of apparently acute strangulation suddenly recover spontaneously; but if the physician is tempted by this rare event to await the result of medical treatment, the case will probably have passed beyond the reach of art. As stated in the last paragraph, the prognosis will mainly depend upon the *early* performance of a surgical operation. If the causes of failure be investigated in operative cases, it will frequently be found to be the result of a septic peritonitis which had been inaugurated before the laparotomy was undertaken. No doubt sometimes the failure is caused by inability to discover the obstructing band, but it must be remembered that the difficulty of finding this and dealing with it is enormously increased by delay.

The aim of the physician, therefore, should be to obtain surgical relief before the virulent bacillus coli has made its way into the peritoneal cavity through the swollen, congested, and inflamed coats of the bowel above the seat of constriction.

Whilst some delay is absolutely essential in order to decide whether or not the case is really one of acute intestinal obstruction, much may be done for the temporary relief of the patient. He must have absolute rest in bed, and collapse-symptoms should be met by a small hypodermic of strychnine

and external warmth. Pain must be relieved, and, since drugging by the mouth is usually inadmissible owing to the vomiting and retarded absorption, morphia hypodermically is about the only drug available. The objection to morphia is its danger in causing such relief to the urgent symptoms as may entirely mask the nature of the case or cause a false estimate of its intensity. The dose, therefore, should be moderate, and not such as will induce sleep ; it should not be too frequently administered, a sufficient interval being allowed to permit the patient to come out of the partial anæsthesia before another hypodermic is administered. Much may be done by poultices or warm or hot fomentations to relieve the patient's suffering. Thirst calls for prompt relief, but usually it is difficult to alleviate this ; ice occasionally relieves, but often does harm by partially filling the stomach with water. A much more reliable method is to administer occasionally or at intervals a large draught of warm water, which will cause a moderately complete washing-out of the stomach.

Lavage of the stomach is a valuable method of treatment in most cases, and gives usually very marked relief ; the soft rubber tube, when tolerated, should be employed ; but, failing this, the administration of large quantities of warm water every four hours may be resorted to.

Food by the mouth should be entirely forbidden ; occasionally a large nutrient enema may be administered. Enemata with the view of washing out the colon are highly recommended by most authorities, but their frequent repetition always does harm ; sometimes in intussusception they may reduce the invagination. A large warm-water enema, if retained, may relieve the thirst considerably. Purgatives, it is needless to say, are rigidly contra-indicated in all acute cases. These measures having been applied, and the signs of obstruction still remaining acute, the abdomen should be opened in the middle line without delay, and any band, cord, or constricting aperture, when found, should be ligatured and divided. If possible, the viscera should not be removed from the cavity of the abdomen. This may sometimes be avoided by opening the most distended and prominent coil after it has been withdrawn from the wound, and the reduction of the general tension thus accomplished may facilitate the discovery of the obstruction.

Volvulus of the sigmoid, when diagnosed, requires as prompt surgical procedure as an internal hernia or strangulation. After opening the abdomen and exposing the distended coil, this should be withdrawn at its most prominent point beyond the lips of the abdominal wound, and incised, with the view of completely emptying it before reduction of the twist is attempted ; the incision in the bowel may be afterwards secured by a double row of sutures. It is necessary, in order to prevent a repetition of the volvulus, to attach the middle

portion of the sigmoid to the parietal peritoneum. It may be necessary sometimes to establish an artificial anus.

Intussusception.—Before resorting to abdominal section, an attempt may be made at reduction by pumping into the rectum air or water after the patient has been fully put under the influence of a general anæsthetic. Sometimes inversion of the body and kneading of the abdominal walls have proved effectual, alone or with copious enemata. Where obvious reduction of the invagination does not take place at once, an abdominal incision should be made without delay and the intussuscepted portion of the intestine most carefully and slowly drawn out of its sheath.

Often reduction will be found impossible owing to swelling and the presence of adhesions ; if small, the entire mass can be excised and the divided ends of the bowel united by sutures. If a large amount of bowel be involved in an irreducible invagination, and if the condition of the patient does not admit of a prolonged operation, there is nothing left for the surgeon to do but rapidly to establish an artificial anus by opening the bowel above the invagination and connecting the intestinal and parietal wounds by suture, trusting to a second operation at a later date for the restoration of continuity. The plan of short-circuiting is very unsatisfactory, and only available in a small percentage of cases.

Strictures may be excised, but if innocent and annular they may be treated when in the small intestine by enteroplasty after a longitudinal incision. Malignant stenosis, when not excisable, may be dealt with by short-circuiting, an anastomosis being established between a portion of the bowel above and below the stricture.

Gall-stones, *foreign bodies*, and *impactions*, when the cause of acute obstruction, are obviously more suitable for expectant treatment than are the foregoing varieties, but when the symptoms are urgent the abdomen should be opened without delay. The foreign body being located, gentle pressure may be exercised upon it by squeezing the intestinal coats behind the obstruction with the intention of forcing it onwards towards the ileo-cæcal valve or anus. If this is not readily accomplished, it may be pushed forwards or backwards into a healthy part of the bowel, which may then be incised and the calculi or foreign substance removed, after which the intestinal incision should be closed by a double line of sutures.

When in a neglected case of strangulation, or other acute obstruction, the patient first comes under observation suffering from all the prostration of a septic peritonitis, with a distended abdomen and stercoraceous vomiting, the question of surgical relief is a very serious one. Any attempt to discover the seat and nature of the obstruction and relieve it by

a radical operation is out of the question. The only available procedure is to perform an enterostomy by first making an abdominal incision and opening the coil of distended bowel which presents itself near the wound, and establishing an artificial anus. Morison recommends an oblique abdominal incision through the external oblique, so as to expose the cæcum. If this be found distended it can be opened ; if not, the nearest coil of distended small intestine should be incised. The operation under such desperate conditions should be done under a local anæsthetic. If the case be one of neglected intussusception, even this palliative operation may not be justifiable, and the patient may be left to the chance of a spontaneous relief through the separation of the gangrenous invaginated intestine.

CHRONIC OBSTRUCTION

The usual cause is *stricture*, simple or malignant, affecting the small or large bowel. The portion of the intestine above the stenosed point becomes in long-standing cases enormously hypertrophied ; the abdominal walls being usually flaccid the hypertrophied bowel in its ineffectual peristalsis is often both palpable and visible, especially during the paroxysms of pain which come on at irregular intervals and are associated with loud borborygmi. This, with the frequent presence of a tumour, renders the diagnosis usually easy and the site of the obstruction obvious

A careful examination of the history will generally afford evidence of the nature of the narrowing, malignant disease revealing itself by the age, wasting, and general cancerous cachexia. Tubercular or dysenteric ulceration, followed by stricture, may be made plain by the history. The constipation is but partial, though in the final stage it may become absolute. It is in the early periods relieved or removed by the use of purgatives, whilst these aggravate the symptoms towards the final stage. Occasionally diarrhœa is present, which may alternate with constipation. In very rare cases the stricture spontaneously may relieve itself by the formation of a fæcal fistula, or even by the establishment of a natural anastomosis ; but a fatal issue is always to be expected once the condition of stenosis has become extreme. This final result is brought about by the supervention of an acute attack with its vomiting and septic peritonitis, some undigested food or fæces having blocked the narrow orifice. Ulceration or gangrene, causing a perforation above the stricture, may suddenly terminate life when the previous symptoms had not revealed the gravity of the case.

Vomiting is never as marked as in the acute cases, and often it is entirely absent till the final acute attack. It is more likely to occur when the stricture is in the small intestine.

When the colon is stenosed, the constipation, though relieved by enemata, does not readily yield to purgatives in the early stage ; it may be absolute for several weeks before death. Often it alternates with diarrhœa, and even when complete in the later stages it may be suddenly and unexpectedly relieved by the spontaneous passage of an enormous amount of fæces. In one case seen by the writer a mass of soft pultaceous fæces was passed which was calculated to weigh no less than fourteen pounds. The evacuation followed a washing-out of the stomach. When the stricture is situated low down in the colon, tenesmus may be prominent, a symptom which never occurs in strictures of the small bowel. The motions in cancer of the sigmoid may present a perfectly normal appearance, as regards shape and size, in the intervals between the attacks of complete constipation or sub-acute obstruction.

The packed colon above the stricture may be mapped out by inspection, palpation, and percussion, unless at the last stage of acute obstruction, when the entire belly may be highly distended and tympanitic. Ballooning of the rectum is nearly always present, but it occurs in other conditions.

Malignant strictures of the colon or small intestines seldom last beyond a year after the first symptoms, whilst the strictures of a simple nature may give rise to symptoms of more or less partial obstruction for many years.

This time-limit in the diagnosis may mislead. The writer once saw in a country district a case of acute obstruction from a cancerous stricture of the rectum which was just within the reach of the finger. The patient appeared almost moribund, but a large motion some hours afterwards relieved the symptoms. More than two years afterwards he found this patient in fair health, but troubled with constipation ; an examination revealed the stricture but little altered. The rectum was removed by the high operation with complete success, and a cancerous stenosis of the true epithelial type was found. Had such a stricture been beyond the reach of the finger, the after-history would have certainly led to a wrong diagnosis. The nature of the growth was verified by a distinguished pathologist.

Fæcal accumulation may occur without stricture in patients suffering from obstinate constipation, and especially in the insane and in aged subjects. The colon may be packed with fæces, though a small motion may be occasionally passed at irregular intervals or even a constant diarrhœa may be the only sign of anything abnormal. When the constipation becomes absolute there may be no escape of any fæces for periods extending over many months. Many cases of total constipation have been reported of a duration of 180 days and upwards.

In all such cases the feculent impaction may be felt through the abdominal parietes as an irregular and usually painless doughy tumour which pits on steady pressure.

Symptoms of acute obstruction may make their appearance at any stage. Sometimes a septic peritonitis supervenes, or ulceration may lead to a rapidly fatal perforation ; more frequently the history exhibits a series of obstructive attacks which pass off with some partial evacuation of scybalæ.

Tumours outside the bowel may cause chronic obstruction by their pressure on either the small or great intestine ; the usual site of growths which act in this way is the pelvis, many of the cases being uterine or ovarian. The constipation is seldom complete till the final stage is entered upon, and the symptoms resemble those produced by stricture. The history of the case and the presence of the tumour seldom leave much doubt regarding the diagnosis.

Adhesions matting together coils of small intestine are often the result of the tubercular inflammation, and sub-acute or chronic attacks of obstruction are often a feature in the clinical picture of abdominal tuberculosis. The symptoms will, of course, vary with the site and with the completeness of the obstruction and consequent constipation, but the constitutional signs of fever, wasting, sweating, &c., alternating often with symptoms of tubercular intestinal ulceration such as diarrhœa and hæmorrhage, usually make the diagnosis clear and eliminate the cases of chronic obstruction caused by other forms of chronic peritonitis and perityphlitis.

Chronic intussusception usually is of the ileo-cæcal type, and may be easily mistaken for the last-mentioned condition, since blood is often present in the motions and emaciation is always well marked. The tumour may often be felt in the cæcal region and its cylindrical or sausage shape may be made out by careful palpation, as the abdominal walls are commonly flaccid. Pain of an intermittent or paroxysmal type usually is present as in the acute invaginations ; but the intervals are much longer, several days intervening between the attacks. The tumour may be found to become harder and more clearly defined during the painful paroxysm.

The bowels are affected very differently, and no rule can be laid down for the recognition of the disease from this point of view. Often they are quite regular, constipated partially or completely, or diarrhœa may be persistent, or all these various conditions may alternate. Blood and mucus very often appear in the motions, and tenesmus is not rare. Purgative medicines may afford transient relief in most cases, and during their action hypertrophied coils of bowel may become evident above the intussusception. Vomiting is never a prominent feature ; often it is absent.

The diagnosis is only clear in those chronic cases which begin as acute obstruction, and often the disease is mistaken for cancer of the bowel, as the patient steadily loses weight and strength and sinks from exhaustion and marasmus. Spontaneous reduction of the intussusception very rarely occurs after the condition has lasted for four or five weeks, and a fatal result is the general rule, often hastened by perforation.

Diagnosis.—The points of value in the differential diagnosis of chronic obstructions have been emphasised in the foregoing brief description.

Prognosis.—This will often be that of the malady which has caused the obstruction, as in cancer, since, the blocking not being complete, the patient may succumb before a fatal acute obstruction sets in. The duration in all the groups of cases is most variable. In simple stricture of the small intestine life in some cases may be almost indefinitely prolonged by rigid dieting. When the colon is the site of the stenosis the issue is always fatal, though life may be prolonged by colotomy. Fæcal accumulation upon the whole rarely causes death, since it yields nearly always to simple treatment, if recognised before ulceration or peritonitis has set in.

Chronic intussusception generally ends fatally if not dealt with surgically.

It must be remembered that even when surgical procedures are impossible the patient may live for half a year after constipation has become established, though ulceration, perforation, gangrene, and peritonitis may supervene at any period and cause a suddenly fatal issue.

Treatment.—The indications for operation not being urgent, the physician has time to watch the progress and symptoms till a fairly accurate diagnosis is possible. The narrowness of the obstructed part of the small intestinal tube may be so great that solid food may bring on sub-acute attacks of obstruction, or, if in the lower portion of the colon, it may only permit liquid fæces to pass. Nevertheless, in either of these cases, should the stenosis be of simple origin, life may be prolonged for very considerable periods by a carefully regulated diet of liquid foods of easy digestibility, and by the judicious administration of laxatives. Every kind of food which leaves much undigested residue must be avoided, fruits and vegetables should be used sparingly, and seeds of all kinds, or jams containing seeds, must be forbidden ; figs are especially bad.

Salol and magnesia must not be given, as both drugs are liable to cause concretions.

Strictures of the small intestine may be dealt with by enteroplasty, by resection of the bowel, or by making an anastomosis between the bowel, above the narrowing, and a coil below it. Sometimes the only measure available is an

enterostomy, but usually an artificial anus in the small intestine ends fatally unless it be very near to the cæcum. When the colon is stenosed, the best procedure is a colotomy close above the obstruction in order to secure immediate relief; and at a later date, if the nature of the case permits of it, a radical operation may be attempted. Even in advanced malignant disease a left colotomy may prolong life for several months. Where the obstruction is high up, the opening having to be made on the right side, the mortality is much greater. Sometimes the bowel may be resected, and an artificial anus left, when the condition of the patient does not admit of a more radical operation, with the permanent closure of the abdominal wound. As in the small intestine, the closure of the colon may be relieved by forming an anastomosis when a radical removal of the diseased area is impossible.

Chronic intussusception may be relieved by an abdominal incision and removal of the invaginated portion, attempts at reduction being usually futile or dangerous.

Fæcal obstruction usually yields to repeated injections given two or three times a day, with the head low and the pelvis considerably raised, the fluid being injected very slowly under a very low continuous pressure. Purgatives are not permissible till the mass is broken up by mechanical means or enemata. An injection of 40 oz. olive-oil often acts most satisfactorily.

In the case of accidental swallowing of foreign bodies, as false teeth, pins, coins, buttons, plum-stones, &c., obstruction of the bowel may be often prevented by a dry diet of stale bread and hard-boiled eggs, which acts by entangling the foreign object and permitting it to be driven by the natural peristalsis towards the anus; whilst purgatives tend to sweep the liquid fæces past the obstruction, and by exciting irregular peristalsis may bring on an attack of acute obstruction. Cotton wool in small amount mixed with the food has been successfully employed.

INTUSSUSCEPTION.

(See under INTESTINAL OBSTRUCTION, pp. 721, 730, 739.)

ITCH. (See SCABIES.)

JACKSONIAN EPILEPSY. (See under EPILEPSY

JAUNDICE, or ICTERUS

This is a condition characterised by yellowish or greenish discoloration of the skin, visible mucous membranes, fluids, and other parts of the body, due to retained bile-pigment, and associated with the presence in the urine of bile-pigment, alone or in company with bile-acids.

Symptoms.—The colour of the skin may vary from a pale citron to a dark olive-green or greenish-brown colour ; in the former condition it may resemble the tint of chlorosis or pernicious anæmia, and in the latter (the so-called black jaundice) it may simulate the bronzing of Addison's disease. The discoloration is usually first and best seen in the conjunctiva ; it is usually well-marked in the mucous membrane of the mouth. Houston and Rankin have observed that the yellowness may be detected in the blood-serum after the clot has separated, long before the skin shows discoloration.

Other skin-disturbances are common. Thus itching is sometimes most distressing in severe cases, and the patient produces various traumatic lesions by the incessant scratching ; urticaria and lichen are common in long-standing cases, and occasionally xanthoma multiplex is present. Petechiæ and large purpuric spots are seen in chronic cases, and boils are not uncommon. Red patches may occur, up to the size of a shilling, formed by bunches of small dilated blood-vessels ; these may be seen over the face and trunk and visible mucous membranes in very chronic cases. The sweat is yellow and will stain linen ; but the tears, saliva, and milk are not usually discoloured ; the bronchial mucus will contain bile-pigment when pneumonia is present.

The urine is always rich in bile-pigment when the jaundice is deep, and it may be almost black in colour. It may be easily demonstrated by placing a small quantity upon a white plate, and causing a drop of fuming nitric acid to come into contact with the periphery of the urinary zone, when the yellow bilirubin is changed into biliverdin and bilipurpurin, with a brilliant play of colours as the two liquids commingle. The test is also easily made by touching a filter-paper, stained by the urine, with a glass rod dipped in fuming nitric acid, or in strong nitric acid to which a crystal of cane-sugar has been added ; the outer zone of green colour is very characteristic, as by it indigogens are excluded.

The urine *markedly stains linen*, and when shaken has a yellow froth which distinguishes it from the discoloured urine sometimes seen after the administration of salol.

Bile acids or salts are usually, but not always, present in excess in the urine ; in some cases of so-called ' blood ' jaundice they may be absent. Their presence or absence is not therefore

of much significance ; they are to be found in excess in many other diseases. Oliver's test is a fairly delicate one : it depends on the power which the bile-salts possess of precipitating peptone ; its application is, however, troublesome.

Hay's test is a rough one ; it cannot be taken as accurate, but it is easily applied by sprinkling a small quantity of sublimed sulphur over the surface of the urine from a jaundiced patient ; the powder sinks at once, but if the bile-salts are not in excess it floats on the surface. This result is due, according to the researches of W. and F. G. Donnan, to the low surface-tension always present in jaundiced urine.

Albumin and tube-casts are commonly present in long-standing cases. Next to the discoloration of the skin and urine, the condition of the fæces is of importance. In all cases where the obstruction of the common duct is complete the bile is prevented from entering the intestine, and the motions are white, resembling pipeclay. The bile is credited with powerful antiseptic properties, which is probably a mistake, as it has already been pointed out that in gall-stone obstruction various microbes flourish in it. Nevertheless, the motions are often very offensive when the bile is absent from the intestine. Fat is sometimes found in the motions, probably owing to the absence of bile, which in the normal state assists in the absorption of fatty materials. Constipation is usually present, but diarrhœa may occur. Walker has pointed out that absence of pancreatic secretion may cause colourless motions, without jaundice being present.

Hæmorrhages are a common complication in all cases of intense and chronic jaundice, owing to diminished coagulability of the blood, epistaxis and purpuric extravasations being common, and sometimes hæmaturia.

The pulse is often markedly slowed, especially in catarrhal jaundice, and the respiration-rate usually is also lowered.

Digestive disturbances and anorexia are common.

Cerebral symptoms, commencing in restlessness, excitement, and delirium, may appear, and pass into coma which cannot be distinguished from that met with in advanced renal disease. This condition is probably independent of the presence of the bile-pigment in the blood, as it is constantly met with in advanced liver-diseases unassociated with jaundice, and it may occur with a normal or sub-normal temperature.

Pathology of Jaundice.—This is a wide and difficult subject, but recent researches tend to simplify it considerably. Formerly all cases of jaundice were divided by hard-and-fast lines into two groups : (1) *Obstructive* or *hepatogenous* jaundice, produced by blocking of the common or hepatic ducts, by gall-stones, tumours, catarrhal inflammation, pressure from without or by strictures ; (2) *non-obstructive* or *hæmatogenous* jaundice, also known as toxæmic, the result of changes in the

blood induced by various poisons. Though this division still serves as a good clinical classification, it must now be accepted in a modified sense in the light of the researches of W. Hunter, which establish the fact that *all jaundice is hepatogenous*, it being certain that the bile is formed in the liver and excreted by this organ. Moreover, Hunter maintains that *all jaundice is obstructive*, the toxæmic or hæmatogenous variety being caused by an increase in the viscidity of the bile, which produces blocking of the smaller ducts. The increased viscidity is itself the result of a catarrhal condition of the lining membrane of the bile-passages, induced by the blood-poison which is excreted in the bile. According to this view, which is based upon advances made in the study of the physiology of the liver, all jaundice is the result of *absorption* of bile from the biliary passages. The bile-pigment is never formed within the blood; hence the acceptance of this truly hepatogenous view of jaundice disposes of the suppression theory that the accumulation of bile-pigment in the blood is the result of arrest of the liver-function.

The most practical classification of jaundice, therefore, is found in still accepting the great class of—(1) *Obstructive* jaundice, confining it, however, strictly to those cases where the blocking is confined to the common bile-duct; and (2) including under the name of hæmohepatogenous jaundice, all those cases where toxæmia or disorders of the blood produce such viscosity of the bile as leads to less obvious obstruction to its flow in the small intra-hepatic ducts.

OBSTRUCTIVE JAUNDICE

1. *Catarrhal jaundice*, caused by acute catarrhal inflammation of the common bile-duct, is a frequently occurring affection in young subjects; its pathology is implied in its nomenclature: the inflammatory swelling of the duct causes obstruction and subsequent absorption of retained bile.

Etiology.—The affection is supposed to originate in some irritation in the duodenum, the result of dyspepsia; hence it is usually associated with or preceded by furred tongue, nausea, and anorexia. Exposure to cold and epidemic causes are operative in some cases, as an epidemic form is occasionally to be met with, and it has been surmised that the catarrh may originate in the smaller ducts, and descend. This is probably the etiology of those cases which occur during pneumonia, the infectious fevers, secondary syphilis, gout, and rheumatism, and as the result of portal obstruction from valvular lesions, &c. Cases of jaundice, following fright and profound mental emotion, are usually included under catarrhal jaundice; Hunter thinks they may be the result of spasm in the smaller ducts. The obstruction is caused by the catarrhal

swelling of the duct at its lower part, usually as it pursues its oblique course through the walls of the duodenum.

Symptoms.—Beyond the gastric disturbances, which are often absent, the first indication of the condition is seen in the yellow tint of the skin and conjunctiva, which is seldom deep, rarely passing beyond a bright lemon-colour. Pain and fever are usually absent, though the latter may be present in epidemic cases.

The urine contains bile-pigment and excess of biliary acids, and the stools are white or clay-coloured.

There is usually a marked diminution of the pulse-rate, which may not exceed thirty-five or forty beats per minute, and the breathing slows in proportion. The liver does not enlarge in mild cases, and the gall-bladder usually cannot be felt ; but in severe cases (which, however, are rarely met with) the liver may be found somewhat enlarged and tender, the gall-bladder may be moderately distended, and there may be some splenic swelling.

The jaundice, in mild cases in children, may pass away in ten days from the onset ; but usually three or four weeks' duration is seen, and sometimes it lasts for six or eight weeks, by which time serious doubts as to the correctness of the diagnosis should arise if the skin-colour is not already showing signs of fading.

The form of jaundice which accompanies hepatic congestion and hepatitis is probably also of catarrhal origin, the smaller ducts being involved.

Diagnosis.—This is usually easily made by the process of exclusion, and the brief duration of the disease without the presence of pain, emaciation, or other serious symptom ; though, beyond the association of dyspeptic symptoms, catarrhal jaundice possesses no distinctive positive symptoms of its own.

Acute yellow atrophy of the liver is a very rare disease, which is not therefore likely to be confounded often with catarrhal jaundice ; the onset is usually, however, associated with marked gastro-intestinal symptoms and some fever. The diagnosis is not possible till the end of the first week, when severe vomiting, hæmatemesis, and delirium set in, which rapidly end in a fatal issue.

Gall-stone jaundice is easily excluded, as a rule, by the total absence of the characteristic paroxysms of hepatic colic.

Cancer of the liver or *ducts* or of the *pancreas* may produce at first identical symptoms in patients past middle-life ; but the absence of a tumour, of marked emaciation, and of ascites are in favour of a catarrhal origin.

Cirrhosis of the liver is, as a rule, readily recognised by the alcoholic history and the jaundice in that disease begins very

gradually and is generally very faint in tinge ; the liver-changes are characteristic, and ascites sets in early.

Weil's disease, or infectious jaundice, begins with severe fever, often with rigors, swelling of the liver and spleen, and albuminuria, and is soon associated with marked weakness and severe nervous symptoms.

2. *Obstructive jaundice* caused by calculi or parasites in the common duct.

This condition has been already dealt with under 'Gall-stones' (page 434). Phillips insists upon the importance of never attributing jaundice to impacted calculi unless there has been *at some time* an attack of hepatic colic ; for though a stone may become impacted without an immediately preceding attack of pain, it is excessively rare for such an event to occur without previous attacks of colic. The danger of diagnosing gall-stones as the cause of an attack of painless jaundice, because of the history of old hepatic colic, must never be forgotten, since cancer so frequently results from the irritation produced by gall-stones which may have been expelled.

All the symptoms and signs of jaundice described upon pages 742–745 are found in their most typical form in this condition ; but if the blocking be incomplete, the jaundice, urine, and stools will afford evidence of this, and fever of the malarial type will supervene.

The symptoms produced by blocking of the common duct with hydatids, or with worms or foreign bodies from the intestine, are identical with those caused by gall-stones.

3. *Stricture of the common duct* occurs after ulceration produced by gall-stones ; the symptoms are a deepening jaundice which is permanent. If the stones have already escaped there will be no colic, and the cause can only be diagnosed when a sufficient time has elapsed for the elimination of malignant disease. Congenital absence of the biliary ducts leads to intense jaundice of the infant before its birth, and it is liable to occur in several instances in the same family, as witnessed by the writer in a family where a number of infants were born without biliary ducts.

The cases are always rapidly fatal if the infant be not stillborn ; they should not be confounded with *icterus neonatorum*. Stricture of the common duct has been recorded by Phillips and others without association with gall-stones, but such cases are extremely rare.

4. Blocking of the common duct by an *ulcer* or *cancer in the duodenum* is comparatively rare. The diagnosis can only be made by recognising the other symptoms peculiar to the primary disease which has caused the jaundice. Thus hæmatemesis, melæna, and some gastric dilatation will be found preceding the jaundice in ulcerative disease ; whilst the

cancerous nature of the blocking will probably produce profound cachexia, wasting, and the formation of a tumour before the jaundice appears. Cancerous and other tumours growing in the duct-walls will produce similar symptoms.

5. Compression of the common duct from *perihepatitis* is usually a sequela of syphilis, and the adhesions which follow as the result of the superficial hepatitis often press upon the portal vein and cause ascites. There is much difficulty in eliminating cholelithiasis, but the history and presence of other tertiary manifestations settle the diagnosis.

6. Compression of the duct by tumours from without may produce intense persistent jaundice : (*a*) The tumour may be in the liver itself, when it will generally be found to be of a malignant nature ; (*b*) it may be in the stomach ; (*c*) in the kidney ; (*d*) enlarged glands at the transverse fissure ; (*e*) in the omentum ; (*f*) in the ovary or uterus ; (*g*) scybalæ in the colon ; (*h*) an abdominal aneurism ; and (*i*), but more frequently, it will be in the pancreas.

Cancer of the head of the pancreas is the most important of all the conditions enumerated in the above list. Phillips insists upon the importance of remembering that, owing to the anatomical position of the gland, jaundice is very liable to occur from blocking of the common duct without interference with the portal vein ; hence ascites is not present unless in the later stages when the disease has extended to other parts, especially when the glands at the transverse fissure become infiltrated. The enlargement of these glands is the cause of jaundice in cancer of the liver, for it is a well-recognised clinical fact, insisted upon by Murchison, that the cancerous mass in the liver rarely blocks the common duct.

In cancer of the head of the pancreas, therefore, the chief symptoms resemble those found in severe catarrhal jaundice or in those caused by gall-stones ; but in the former case the discoloration passes away in a few weeks, and in the latter there is severe colic. The condition of the gall-bladder is a matter of great diagnostic value, as insisted upon by Mayo Robson. It is seldom found as a palpable tumour in catarrhal jaundice, and even in gall-stone cases it is usually either not to be felt or, if an obvious tumour exist, it is usually of moderate dimensions. In pancreatic cancer, however, along with the intense jaundice, the gall-bladder is usually greatly distended, and remains so. The presence of a hard nodular tumour in the region of the pancreas, and an unusual degree of emaciation, especially if these precede the jaundice, are of great diagnostic value ; the tumour may convey the pulsations of the aorta to the hand of the examiner. Pain, when it is present, is of the severe continuous type, aggravated by coughing, and does not occur in the typical paroxysms of hepatic colic, though it must be remembered that gall-stones may possibly be

associated with pancreatic cancer. Fat may in rare cases be found in the stools, and the other signs of arrested function of the pancreas may also exist; but these never appear till the disease has been of considerable standing, or has caused such an amount of interstitial pancreatitis from secondary cystic disease as will affect the islands of Langerhans and cause glycosuria.

HÆMOHEPATOGENOUS JAUNDICE

Etiology and Pathology.—This corresponds to the old class of non-obstructive or toxæmic jaundice. As already stated, the discoloration is caused by obstruction in the small ducts, which is the result of a very viscid condition of the bile. This viscosity is produced by some poison in the blood which, upon being excreted by the liver, so irritates the biliary passages that the bile becomes greatly altered in character and the epithelial lining suffers injury, though there is no obvious blocking of the large ducts. This form of jaundice can easily be produced by toluylendiamin, phosphorus, arsenic, and other chemical poisons. It occurs as part of the clinical picture of yellow fever, malaria, and snake-bite, and it is sometimes met with as a complication which marks a serious blood-poisoning, in septic conditions, and in most of the infective fevers. It is the obvious feature in acute yellow atrophy of the liver and in Weil's disease, and is probably the explanation of icterus neonatorum.

Symptoms.—Hæmohepatogenous jaundice may be differentiated from jaundice the result of blockage of the common bile-duct by the following characters. Usually it is not so intense, rarely approaching the dark olive-green seen in gall-stone cases. The constitutional symptoms are generally much more profound, varying with the nature and intensity of the poison which has produced the blood-changes; hence epistaxis, black vomit, delirium, and coma are often found associated with the discoloration of the skin. Renal symptoms, as albuminuria and suppression, are common towards the termination of the disease.

The urine from the commencement of the jaundice shows a marked difference from that seen in the obviously obstructive cases; it does not exhibit increase of the bile-acids; in some instances the bile acids or salts may be altogether absent. The urine usually contains bile-pigment, but its dark colour may be chiefly owing to the ordinary pigments.

The most obvious difference, however, is apparent upon examination of the stools, since these will usually contain much bile, if not constantly, certainly in all cases at frequent intervals during the progress of the case, whilst the pipeclay stools met with in the obstructive forms of jaundice are not seen.

Cases of severe hæmohepatogenous jaundice sometimes occur in epidemics, constituting the so-called 'infectious' jaundice which appears to be identical with or closely related to Weil's disease. They are also occasionally met with sporadically, and are then known as *icterus gravis*, or malignant jaundice, and these bear a close resemblance to acute yellow atrophy. Outbreaks of icterus gravis have been frequently reported, and by some authorities they are regarded as severe examples of Weil's disease; whilst others employ the terms 'malignant jaundice' and 'icterus gravis' as synonyms of acute yellow atrophy of the liver. It is probable that all these forms of disease are instances of profound blood-poisoning caused by the toxins produced by infective bacteria; whether the micro-organisms are the same in each type of jaundice is a matter which is yet to be settled.

Infectious or *febrile jaundice*, as described by Weil, is an acute infectious disorder characterised by fever, splenic enlargement, albuminuria, jaundice, and severe muscular pains. It occurs most frequently amongst young male adults of the labouring class in hot weather, being especially prone to attack slaughter-house workers and sewer cleaners.

Symptoms.—The fever commences abruptly, and reaches 103° or more in forty-eight hours; the pulse is correspondingly raised; marked headache, with usually smart delirium, is an early symptom. Muscular pains of a severe type affect the legs and compel the patient to take to bed; they may involve all the muscles of the body, including those of the face. Jaundice appears after the second day; the liver swells and is very tender, and the splenic enlargement is palpable from the onset of the attack. Albuminuria, and even hæmaturia, may appear before the jaundice. The stools are usually somewhat lighter than normal in cases where the jaundice is severe.

In about five or six days the symptoms gradually commence to improve and the fever slowly disappears, but it is liable to return after a complete intermission of five or seven days. Convalescence is slow; the disease is only fatal in a small percentage of cases. The cause of the disease has been found by Jaeger to be a bacillus, which he has named *proteus fluorescens*.

Jaundice of new-born infants, or *icterus neonatorum*, usually appears about thirty-six hours after birth. It is seldom intense, and usually passes off in a week or ten days or less. It is doubtful whether it should be classed as a simple obstructive jaundice, produced by blocking of the medium or larger ducts, or whether it be a true toxæmic affection originating in some blood-changes from faulty hygiene, as it is very common in foundling hospitals. Some authorities regard it as the result of an increased destruction of the red blood-discs. It certainly appears to occur in those cases where the infant's

face is enveloped in clothing which interferes with the due aëration of the blood.

It should not be confounded with the rare and interesting type of infantile jaundice which depends upon absence or malformation of the biliary ducts, already referred to, or with those cases of jaundice arising from sepsis through the stump of the severed umbilical cord.

As a rule it may be said that the prognosis and treatment of the different varieties of jaundice are those belonging to the primary affection.

KELOID, or CHELOID

A condition has been often observed when surgical wounds heal by granulation-tissue, the newly formed fibrous material taking the form of fleshy welts or excrescences of scar-tissue, which may extend by a series of claw-like processes into the surrounding skin, forming elongated tumours which cause considerable deformity.

When the cord-like thickening is confined to the immediate site of the original wound, the condition is usually termed an hypertrophied cicatrix. In either case the fibroid growth is confined to the corium, and appears first as a pinkish swelling, upon the surface of which dilated vessels often appear. Morris has shown that the microscopic appearances of the connective tissue consisting of collagenous bundles indicate a pathological process which may be regarded as a connecting-link between ordinary scar-tissue and sarcoma. Neighbouring indurations may coalesce, forming irregular-shaped ovoid plaques, which, if they throw out claw-like processes, may involve a considerable area of the skin ; finally increasing in density, they may become of almost bony hardness, though still retaining their smooth surface and pink colour. In this condition they may be very tender or painful, but they very seldom, if ever, break down or ulcerate, and may remain permanent or undergo gradual involution, especially in young subjects.

The condition has often been met with in cases devoid of any history of a wound, and then it has been described under the names of Idiopathic or Spontaneous Keloid, True Keloid, or Keloid of Alibert. Most authorities now regard all examples of the disease as secondary or acquired, originating in scar-tissue, since the most trivial abrasions—as those produced by scratching, leech-bites, herpes, acne, syphilis, lupus, small-pox, boils, psoriasis, &c., as well as the injuries produced by burns and severe wounds—may all be followed by the characteristic connective-tissue outgrowths. Some authorities regard keloid as originating in a local tubercular infection, and

Legrain succeeded in producing local tuberculosis in the rabbit by inserting a fragment of keloid tissue from a scar under the skin of the animal. In this case it must, however, be noticed that the scar-tissue was taken from a tuberculous source.

The most common seat of keloid unassociated with obvious injury is over the sternum or some part of the trunk, face, or head, and it is often multiple when following such affections as small-pox and pruritus.

Diagnosis.—The scar-like appearance and the slowly growing radiating prolongations form a clinical picture easily differentiated from the more rapidly formed cutaneous *sarcomatous* and *cancerous* tumours of the skin. In rare cases it may be associated with warty growths which might be mistaken for *lupus verrucosus*, but the history of its originating in a wound will prevent an error in diagnosis. Keloid, as above described, must not be confounded with the so-called 'true cheloid of Addison,' which is the name unfortunately sometimes applied to sclerodermia.

Treatment.—This is often unsatisfactory. Excision of the scar-tissue is nearly always followed by a return in the new cicatrix, and especially if caustics have been employed. Deep scarifications which divide the vessels supplying the growths are often effectual when repeatedly performed. Electrolysis, in the hands of Morris, combined with daily massage, has given good results. A most effective agent is elastic pressure, and in one long-standing case in the writer's experience a large keloid growth over the tibia was rapidly caused to disappear under the pressure of a rubber bandage. Recently excellent results have been obtained by Finsen's light and X-ray treatment. The writer has seen numerous examples of extensive keloid growths which so often follow the new radical operation for the removal of lymphatic glands in the neck, all of which have disappeared spontaneously in the course of a year or two.

KERATOSIS

This is the name given to several skin-conditions in which there is the common symptom or sign of a considerable accumulation of the horny cells of the epidermis. Thus, in the milder forms of ichthyosis, characterised by the nutmeg-grater variety of skin, the condition is termed Keratosis Pilaris. The horny cells form papules which are in no way associated with inflammatory action, and these papules plug the orifices of the fine hair-follicles on the extensor aspect of the limbs, each minute elevation being often built round a small hair-shaft, the entire papule being easily scraped off. This condition is described by some dermatologists as *Lichen*

pilaris. It may occur in filthy subjects who are not affected with ichthyosis, and it can be distinguished from a true inflammatory lichen pilaris by the absence of the changes indicative of inflammation. The treatment suitable in the mild forms of ichthyosis effects its speedy removal. A form of keratosis is frequently observed in chronic arsenical poisoning, the hands and feet being the usual sites. It is easily removed by salicylic paste.

Keratosis follicularis is a rare and probably contagious disease of the skin also known as 'Darier's disease' and 'ichthyosis sebacea cornea' (Wilson), which is believed to be the result of infection with psorosperms of the coccidia class.

The disease may invade any portion or the entire cutaneous surface; its chief site is the hypogastrium, groins, and perinæum, but it may involve the face and scalp. It begins with isolated papules which are capped by a brownish-yellow horny crust, elevated considerably above the level of the skin. These can be detached from the underlying skin to which they adhere, their removal leaving a small pit filled with softer material, which can be squeezed out by the pressure of the finger-tips; these little depressions are the dilated orifices of sebaceous glands. The plug of sebum removable by squeezing has been found to contain oval or spheroidal highly refractile cells believed by several authorities to be psorosperms, whilst other observers have satisfied themselves that these bodies, or 'grains' as they are called, are but degenerate epithelial cells, because they resist both acid and alkaline reagents, and as yet have not been proved capable of inoculating animals. The crust-topped papules join with neighbouring ones, forming nodular masses of various sizes, with much thickening of the skin, which may become fissured and moist by decomposing fetid secretion.

The disease, though affecting the health but slightly, tends to spread, and may ultimately cover the entire body.

The treatment of the affection is most unsatisfactory, the only agent of marked value being the early removal of the papules as they appear by the thermo-cautery, as practised by Schuimmer. A paste of salicylic acid, used as in lupus verrucosus, has been employed in the later stages with some benefit.

KIDNEY-DISEASES

Acute and chronic affections involving the tubules, glomeruli, and interstitial tissue of the kidney have been described under 'Bright's Disease.'

Amyloid disease, owing to its frequent association with the other forms of renal affection, is also described under 'Bright's Disease' upon page 129.

Hydronephrosis and Pyelitis are described under their respective headings, the latter term including such conditions as pyonephrosis, consecutive nephritis, and pyelonephritis. Perinephritic abscess, for convenience of reference, will also be described under 'Pyelitis.'

The remaining diseased conditions of the renal organs will be here described. They are cystic disease, floating or movable kidney, stone, and tumours.

CYSTIC DISEASE OF THE KIDNEY

Small cysts are often found in a kidney which is the seat of granular disease; they are probably the result of dilatations arising from a blocking of uriniferous tubules, or of Bowman's capsules containing the Malpighian tufts. They occur in the wasted cortical portion of the organ and are visible on its surface; they are of no clinical interest except in the very rare cases where they may be felt through the abdominal walls.

Simple serous cysts are not uncommonly met with in the kidneys of old people without granular or other form of renal disease. These likewise are cortical, and probably the result of obstruction. When numerous and large, by their pressure they may cause atrophy or wasting of the renal cortex. The fluid in their interior usually is devoid of urea, but may contain albumin and blood; more commonly it is a very dilute colourless or amber saline solution. This form of cystic disease may reach to considerable dimensions, but very rarely does it ever attain the colossal proportions seen in the variety of the disease to be next mentioned. It may, however, increase to an extent which may simulate hydatid disease, hydronephrosis, malignant disease, or ovarian tumours. In the latter case the diagnosis may be most difficult, as some of these rare cystic growths may contain gelatinous or colloid material.

General cystic disease is almost invariably bilateral and probably always congenital, though the disease may not be recognisable till adult life. In this form of renal disease the entire organ is the seat of a large number of cysts which vary in size from that of a large pea to a Tangerine orange. This enormous mass of conglomerate cysts may fill up and greatly distend the abdominal cavity of the fœtus, so that delivery may be rendered most difficult, and in adult life the kidney most affected may weigh as much as seven pounds. The individual cysts do not communicate with each other: they are separated by fibrous bands containing the remnants of the atrophied medullary and cortical secreting structure; each cavity is lined with a layer of flattened epithelial cells and contains a clear amber-coloured, slightly albuminous liquid, holding in solution traces of salts, but rarely urea or uric

acid. Occasionally, however, the contents may be turbid, viscid, colloid, or bloody, and triple phosphates and cholesterin are sometimes present.

Etiology and Pathology.—The origin of these cysts is obscure. Some regard them as produced by dilatation of uriniferous tubules, or Bowman's capsules, as in the case of the cysts in contracted kidney ; others, notably Shattock, explain their existence on the hypothesis that they are due to aberrant development of the mesonephron, or Wolffian body. In the early fatal cases other abnormalities in development, as talipes, hare-lip, cystic disease of the liver, imperforate anus, &c., have been found present ; and Osler has recorded the interesting fact of a mother and son being the subjects of the disease. The view of the congeries of cysts being a conglomeration of small adenomatous growths (Bland Sutton) or mucoid endotheliomata (Courbis) is advanced. It is held by some authorities that the cystic disease met with in adults is an acquired disease, differing from the congenital variety, both conditions being the result of dilatation of tubules by uric-acid infarcts (Virchow). The ureter is usually normal or somewhat narrowed, and the pelvis of the kidney may be considerably dilated.

Though the renal organs can scarcely be recognised as such when removed from the body, the secreting structure being merely represented by shreds of tissue between the cysts, nevertheless the entire conglomeration of these usually retains the reniform outline or configuration, and the cystic degeneration is commonly much more advanced on one side of the body than on the other.

Symptoms.—Unless in an advanced stage of the disease, symptoms may be entirely absent, but ultimately the clinical features of a chronic interstitial nephritis develop. The urine is increased in quantity, is pale, and of low specific gravity, and contains a small amount of albumin, with hyaline and granular casts. Hæmaturia often makes its appearance at irregular intervals. There is almost always some evidence of hypertrophy of the heart, and high arterial tension caused by peripheral resistance. Pain in the loins and dropsy of the legs are common from the pressure of the enlarged organs ; and death usually occurs from uræmia, unless hastened by hæmaturia or cerebral hæmorrhage.

The chief diagnostic sign is the presence of a tumour in each lumbar region, and when this can be palpated the case is clear if the symptoms just mentioned are present. Though this rare disease is said to be much more common in men than in women, the only two examples seen by the writer were in women. In one the growths were so large that the cystic tumours met in the middle line, forming a swelling which reached from one loin to the other, occupying the entire

upper part of the abdomen immediately below the liver, which organ was possibly also cystic.

Diagnosis.—The simple cysts which spring from the cortex may be few in number, any one of which may, however, become large enough to form a soft fluctuating tumour which can only be distinguished from an hydatid cyst after tapping. In the multiple or poly-cystic form the enlargement is more uniform, since few of the cysts exceed an ordinary-sized marble in bulk ; the entire tumour will move but little in inspiration, and though it may feel soft to the touch it will not fluctuate. It may preserve on both sides to some extent the reniform shape or outline; on either side the large intestine will be in front of it, and on the left it will be overlaid by the stomach. The flanks will bulge a little, but the tumours do not project backwards. The palpation of the growth by placing one hand behind between the last rib and the iliac crest, and the other hand over the corresponding part of the abdominal wall in front, whilst the patient lies flat and makes a prolonged expiration, will usually reveal the cystic enlargements when the fingers are made to approximate or alternately to cause pressure and relaxation. A varicocele has been found when the tumour is very large and on the left side.

Prognosis.—The smaller cysts are of no consequence save from their mechanical effects. In the true cystic disease of the kidney the prognosis is always grave. It is very grave when the physical signs show that the tumours are steadily increasing, and death is not far off when symptoms of renal inadequacy begin to show themselves. A prognosis as regards time may be impossible ; this is evident if one accepts the theory that all the cases are of congenital origin. Assuming that this is the fact, some assistance in forming a forecast may be obtained by watching the tumours and carefully examining the composition of the urine, and estimating the output of urea and the excretory quotient, as recommended by Wright. A period of life of five or seven years from the time that the disease is first diagnosed will rarely be exceeded. This period may at any time be suddenly brought to a termination by a profuse hæmaturia, uræmic convulsions, apoplexy, œdema of the lung, or pneumonia.

Treatment.—Simple cysts, when they become large enough to produce mechanical effects, may be tapped, and after refilling, should this occur a few times, they may be freely laid open and the cyst-wound attached to the edges of the skin-incision for drainage.

General cystic disease of the kidney must, however, be dealt with surgically only after serious consideration, since the disease may be practically regarded as always bilateral, and the excision or removal of one organ is sure to be speedily fatal if the other is much involved.

Some information may be obtained by collecting the urine from each ureter. Several instances are on record where the patient has survived many years after the removal of a large cystic kidney. In most cases the question of treatment resolves itself into that of chronic interstitial nephritis, the main indication being steadily to maintain vicarious elimination by the skin and bowel and keep up the condition of cardiac compensation.

Hydatid cyst of the kidney has been dealt with on page 657.

KIDNEY, DISPLACEMENTS OF

Under 'Enteroptosis' or 'Glénard's Disease,' so-called, the displacement of the renal organs has been incidentally referred to, since the dislocation of one or both kidneys is often a part of the general abnormal condition of relaxation.

The degree of displacement varies considerably from that in which the kidney is said to be merely *palpable*, when its lower third can be felt by the fingers, to that in which it exhibits a degree of mobility which enables the physician to grasp it and to freely move it about a considerable portion of the abdominal cavity, when the condition is usually termed *floating* kidney. An intermediate degree of mobility is common in which the organ may be hooked down by the fingers applied above its upper extremity so that the entire anterior surface may be palpated by the fingers of the other hand; to this condition the term *movable* kidney is commonly applied.

The term 'nephroptosis' is often loosely applied to all degrees of mobility of the kidney.

The normal position of the kidneys, according to Symington, is behind the peritoneum, the greater portion of each being situated in the hypochondriac and epigastric regions, the lower ends encroaching upon the lumbar and umbilical regions, the right kidney being a little lower than the left. The organs are maintained on a level with the twelfth dorsal and the first two or three lumbar vertebræ by their vessels and loose capsule of areolar tissue, which contains much fat, and also by the tonicity of the abdominal muscles, which keeps up a certain degree of general hydrostatic pressure in the abdominal cavity. The most effective agent, however, in maintaining the organ in its normal position is the band of peritoneum which crosses the upper part of the right kidney and separates it from the renal fossa on the surface of the liver; this support it receives from the reflexion of the membrane covering the commencement of the transverse colon. The lower end of the right kidney also receives upon its anterior surface some peritoneum, as the membrane extends from the

right colon to the adjoining third part of the duodenum. On the left side the peritoneum is in a somewhat similar manner reflected from the descending colon to the anterior surface of the kidney, which is, moreover, transversely crossed by the pancreas and splenic vessels about the level of its hilum ; from the borders of the pancreas it receives some additional support from the peritoneal attachments, as pointed out by Landau.

The minor degrees of mobility are accounted for by the laxity of the areolar tissue, and the disappearance or diminution in the amount of fat in the loose capsula adiposa, as well as by some dragging upon the peritoneal reflexions, which conditions may permit of the organ slipping up and down in its loose capsule, or, as pointed out by Macalister, the kidney and its capsule may move up and down behind the peritoneum. In the case of a floating kidney a more or less complete mesonephric fold of peritoneum enables the organ to move freely in obedience to the laws of gravity or in the directions of force received from neighbouring viscera.

Hurry Fenwick maintains that the chief etiological factor in movable kidney is the contractions of a full cæcum in its efforts to drive upwards the intestinal contents. In doing so it drags upon the cord-like band described by Longyear, which extends behind the peritoneal attachment from the framework of the fatty capsule at the lower pole of the kidney to the posterior surface of the ascending colon as far downwards as the ileum.

During life there is evidence of slight changes in size and position in the healthy kidney, which, however, bear no appreciable relation to the pathological mobility under consideration ; though after death, owing to the increase in the solidity of the fat, the kidney usually appears to be more firmly fixed.

Etiology.—As already stated, nephroptosis is often but a part of the general ptosis described by Glénard, and the causes which are operative in the induction of that condition maintain in effecting the renal mobility. These are relaxation of the abdominal walls, especially in association with successive pregnancies, tight-lacing, violent straining, lifting of heavy weights, or the continual bearing about of heavy clothing attached to the waist. All of these causes may, however, produce mobility of the kidney without any general ptosis. Chronic and obstinate constipation, by which a distended colon may drag upon the peritoneal covering of the kidney, is undoubtedly a common factor ; and alone, or in association with this, obstinate vomiting or severe retching, as in sea-sickness, may be easily understood to be an efficient factor. Rapid emaciation, which removes the fatty padding from the loose areolar capsule, answers for some cases. The question whether the presence of a distinct mesonephron is of

congenital or acquired origin is difficult of solution ; probably some degree of congenital relaxation of the peritoneal attachments or elongation of the vessels may have been present as predisposing causes in most cases of freely movable or floating kidney.

It occurs about seven times more frequently in women than in men, and somewhat more than seven times on the right side for once on the left; and in about one-seventh of all cases both kidneys are affected. The frequency of right nephroptosis is explained by the proximity of the right kidney to the liver ; but it appears to be more likely due to the support which the left organ receives from the peritoneal attachments, passing between the outer renal capsule and the upper and lower borders of the pancreas. The greatest number of cases in women occur about the middle period of life, a few only in those who have not borne children, and the condition is rare in childhood.

Symptoms.—In a small majority of all cases there are no symptoms ; in other words, the condition in a little over half of the total is discovered accidentally by the physician or the patient. The signs indicative of nephroptosis are not easily mistaken if due care be exercised in the examination. The patient should lie upon her back with the knees bent; if she be a small thin subject the physician grasps her flank by placing his fingers behind over the eleventh and below the twelfth rib, with the thumb resting upon the soft tissues below the costal arch in front. A movable or palpable kidney can thus be grasped on the right side between the fingers and thumb of the physician's left hand ; it may be felt to slip up a little during expiration, and downwards during inspiration, or it may be made to move independently of the respiratory act by substituting the finger-tips of the right hand for the left thumb and hooking the organ down as far as the level of the umbilicus. In the case of a floating kidney it may be easily pushed across the middle line or downwards as far as the brim of the pelvis ; its reniform outline may be also distinguished, and usually it is free from tenderness. In thin relaxed subjects it may be grasped by the fingers and thumb of one hand like a tablet of soap between the folds of a towel, when, if steady, firm pressure be applied, a sickening and decidedly painful sensation will be complained of by the patient. Bonney advocates examination when the patient is in the erect posture, the loss of the expiratory return or ascent of the organ being recognised by palpation, which will also demonstrate the oblique or transverse position of the movable organ.

The commonest symptom of movable or floating kidney is a painful dragging sensation, as if a weight were pulling something downwards from the hypochrondriac region—a sensation

which is usually aggravated at the menstrual period, when it may become neuralgic in its intensity. Hysterical manifestations are common, especially in those cases where the nephroptosis is part of a general ptosis of the intestines and other viscera. An hysterical patient who discovers or has been informed of the presence of a floating kidney very soon develops marked pain and tenderness in the movable organ. In males hypochondriacal symptoms are liable to supervene, and they often complain of pain along the spermatic cord, in the inguinal canal, or in that part of the thigh supplied by the crural branch of the genito-crural nerve, which passes beneath Poupart's ligament on the surface of the psoas muscle.

Dyspeptic symptoms are very often present; these are either of the nervous forms met with in hysteria, or are due to dilatation of the stomach secondary to its ptosis, which is often a part of the general enteroptosis. It is, however, highly probable that the dragging of a pathologically dilated stomach may sometimes pull down the kidney from its moorings. It is supposed also that the movable kidney may cause retention of the stomach-contents and dilatation of the organ by pressure on the duodenum—a condition which may also account for the violent gastric crises sometimes met with. Macalister, in his able monograph in Allbutt's 'System,' describes how the duodenum becomes obstructed by bands passing between it and the movable kidney, which explains the temporary jaundice and dilatation of the gall-bladder sometimes observed. Torsion of the renal vein and ureter sometimes gives rise to symptoms of great severity (Dietl's crisis). The writer has seen a case in an aged multipara where a floating kidney had fallen down into the pelvis, causing intense bladder-tenesmus and agonising pain, which was made instantly to disappear on complete inversion of the body. In another case an attack of uncontrollable vomiting was similarly relieved.

Hydronephrosis sometimes follows, and occasionally the movable organ becomes atrophied owing to the interference with its blood-supply. The urine shows little change, but sometimes intermittent polyuria coincides with a considerable reduction in the bulk of the floating tumour. Hæmaturia is rarely observed, but it has been noticed to follow severe attacks in which the ureter or renal vein had become twisted or kinked.

Extreme emaciation and obstinate constipation sometimes occur sufficient to warrant a diagnosis of malignant disease.

Diagnosis.—The signs already described are usually sufficient to prevent mistakes; but it must not be forgotten that often the symptoms bear no proportion to the mobility of the organ. The effect of position is an important element in diagnosis; the dragging pain, vomiting, &c., often

disappearing upon assuming the horizontal position ; and in some instances, as already mentioned, inversion of the body may be tried.

Ovarian tumour can hardly be mistaken for floating kidney, except in stout patients where the outline of the growth cannot be distinctly made out. An ovarian cyst must be very small to be mistaken, and if care be exercised in the examination it will be found impossible to move it up into the normal position of the kidney, owing to the shortness of its pelvic attachments.

Pyosalpinx will be differentiated in a similar manner, and *hydatids* of the liver or other organs, unless very freely movable, are not liable to be confused ; but the distinct fluctuation and the nature of the hydatid fluid removed by the hypodermic needle are conclusive.

Tumours of the bowel may simulate a floating kidney, but the smooth outline and the sickening sensation caused by tightly grasping the latter are very distinctive ; a bowel-tumour or glandular growth in the mesentery never presents to the physician's grasp the smooth renal outline and uniform, firm, elastic sensation.

Enlarged gall-bladder, though it may be freely moved from side to side, can never be forced down to the pelvis. It is pear-shaped, with its apex upwards, and can never be grasped between the fingers and thumb in the way a floating kidney can, though it may be so close to the abdominal wall as to be visible, following the liver up and down in respiration. On distending the colon with gas, a movable kidney is made to disappear into its bed, whilst a distended gall-bladder is pushed forwards and becomes more evident.

Splenic enlargement is differentiated by its outline and notch and by never having the bowel in front of it.

It must not be forgotten that a *stone* or *growth* in the kidney may be the cause of its mobility or ptosis, in which case the urinary characters, taken with the history, usually afford evidence for conclusive diagnosis.

Treatment.—Though the condition is usually quite harmless and does not tend to shorten life, treatment is sometimes urgently demanded for the relief of acute suffering.

Palliatives in the form of prolonged rest, or bandaging over a suitable pad, applied when the patient is in the horizontal position before getting up in the morning, should be tried. Various forms of abdominal binders with pads, springs, or pneumatic trusses, have been devised. Almost any appliance which increases the pressure inside the abdomen will keep some movable kidneys in place, whilst others are influenced injuriously by every attempt at external abdominal support. The value of sudden inversion of the body for the relief of urgent suffering has been already referred to. It may be

easily accomplished by placing the patient on a couch, which can then be tilted up to any angle.

Prolonged rest, with over-feeding, may prove curative in some cases dependent upon emaciation.

Surgical procedures are resorted to when the symptoms are severe and palliatives fail. Nephrorrhaphy, or stitching of the organ to the abdominal wall, has proved a successful operation in numerous cases. The operation, as practised by Fullerton, who suspends the kidney by a piece of its capsule from the ligamentum arcuatum externum, has given excellent results in his hands. Nephrectomy is a more serious operation, and should only be advocated when the movable organ is diseased and the opposite kidney quite sound.

KIDNEY, STONE IN

This condition, which is known as Nephrolithiasis, is one caused by a deposition of the urinary salts in a uriniferous tubule, in one of the calyces or in the pelvis of the kidney.

The calculus is usually built around some small nucleus of organic matter, as a cluster of bacteria, tubal epithelium, blood-clot, &c. The feeble solubility of uric acid, especially when the urine is highly acid, explains the greater frequency of uric acid calculi. Oxalate of lime (the mulberry calculus) follows next in frequency, though it is commonly associated with uric acid. Urate of ammonium is found to make up the bulk of the calculi met with in young children and infants.

Phosphatic calculi are common, the phosphate of ammonium and magnesium being sometimes blended with the calcium phosphate; these may appear deposited in alternating layers on a uric acid or oxalate of lime calculus. Calcium carbonate, xanthin, cystin, and other forms are rare. The stones may be numerous or solitary, and of all shapes and sizes; the so-called renal sand or gravel consists mainly of uric acid or urates which, owing to some error in metabolism, are deposited in showers of crystals, some of which coalesce, forming particles as large as a hemp-seed, which may be discharged for long periods without causing pain in their passage down the ureter.

When a renal calculus fails to enter the ureter, it may continue to increase in size by the deposition of urinary salts as it lies in one of the calyces or in the pelvis of the kidney, until it forms a complete mould of the renal pelvis. Little trouble may be caused by such a coral-like stone, though of large dimensions. When, however, one of its projecting arms or branches blocks the entrance of the ureter, the secretory function of the kidney becomes seriously interfered with, so that changes of an atrophic nature in the uriniferous tubules

and glomeruli occur. Any stone which blocks the entrance of the ureter is liable to cause more or less hydronephrosis; but this may ultimately disappear, and the final change be an atrophic cirrhosis of the organ.

A stone may be dislodged from its bed in one of the calyces or in a corner of the renal pelvis in some violent movement of the body, when considerable hæmaturia may result from injury of the tissues, and pus may form, causing renal abscess or perinephritic suppuration. It is, however, unlikely that these latter changes occur without the introduction of septic organisms, which find their way from the urine through some abrasion of the epithelial lining of the pelvis of the kidney.

In this way, also, an old hydronephrosis of intermittent character may become converted into a pyonephrosis.

The calculus may be single and confined to one kidney, or there may be a number of stones, and occasionally both glands are the seat of concretions.

No age is exempt from renal calculi; males suffer more frequently than females, and the condition is much more frequent in some localities than in others. Whether this be the result of some endemic causes which modify metabolism, or whether the composition of the drinking-water is to blame, or whether both factors may be operative at the same time, are questions not easily settled. Sometimes one calculus is formed and expelled, and the patient is never afterwards affected; whilst in other cases stones are continually being manufactured and passed at irregular intervals for many years.

Symptoms. — As a rule, the patient is unaware of anything being wrong till the calculus enters the ureter, when, if it be of sufficient size, an attack of renal colic is produced suddenly. This may commence with a rigor, and vomiting is not unusual. The predominant feature is pain, which is of a severe paroxysmal nature caused by the contraction of the muscular fibres of the duct in the effort to drive on the concretion, and it is never, as sometimes erroneously stated, caused by the pressure of the urine behind the obstruction; no such pressure exists. The pain darts along the course of the ureter and lumbar nerves, being felt in the flank, inside of the thigh, and in the testicle; sometimes also it is referred to the head of the penis. In severe agonising cases the pain may radiate beyond the usual sites and be felt in the back or chest.

The duration of the attack varies as greatly as its severity. Sometimes a small smooth stone is rapidly driven into the bladder in a few minutes with an abrupt termination of all suffering. At other times several hours of intense agonising suffering, consisting of rapidly succeeding paroxysms of pain, are experienced, which may almost entirely fade away for

long or short periods, suddenly to deepen into acute exacerbations. During such prolonged attacks there may be serious constitutional symptoms of collapse, with feeble and rapid pulse, vomiting, sweating, and fever.

Usually the bladder participates in the disturbance, micturition being frequent, painful, and often bloody ; but sometimes very large quantities of clear limpid urine are painlessly voided, probably owing to over-activity of the unaffected kidney. In rare cases there is total suppression of urine, which may give rise to the suspicion that the opposite ureter had been previously blocked, though such anuria is usually of short duration.

After the cessation of the paroxysms a dull pain may be felt for many hours in the region of the kidney and along the course of the ureter. The attack is often brought on by some severe exercise or shaking, though no such exciting cause may be forthcoming in other cases.

When a stone has previously blocked the opposite ureter and caused destruction of its secretory function, total suppression of urine will result till the recently formed calculus finds its way into the bladder. Should this, however, not occur, death follows ; but the fatal result may be delayed for ten days or even for a fortnight, marked symptoms of uræmia only supervening after many days of complete anuria. The pupils are contracted ; there is slow laboured breathing, twitching of the muscles, sub-normal temperature, restlessness passing into drowsiness, vomiting, and thirst ; but coma and convulsions do not supervene as in ordinary uræmia.

In thin subjects the stone may be felt through the abdominal parietes somewhere in the course of the ureter, but such an event is rare, for two reasons—a stone must be of small dimensions to travel down the duct, and the tenderness and rigidity of the muscles usually prevent the necessary palpation.

The symptoms produced by a stone which remains in the kidney vary considerably, and, as already remarked, they may be entirely absent. Ultimately, however, hæmaturia is liable to appear, especially after some severe shaking of the body, as in jumping, or riding in a jolting vehicle. Pain in the kidney is generally felt in association with the appearance of blood in the urine. The pain is usually aggravated by movement, and though felt chiefly in the lumbar region it may radiate widely, and has even been mistaken for sciatica. It is of a dull aching character in most cases, but occasionally it comes on in paroxysms so severe as to simulate the typical renal colic. Even when always dull and constant, the pain may be referred to the regions affected when a calculus is passing down the ureter. Thus it may be felt in the testicle or inside of the thigh, and even along the inside of the leg as far as the knee or foot. Cases have been recorded where the

pain produced by a large stone in the kidney has been transferred to the loin and renal region on the sound side.

Tenderness is nearly always found when sought for, and is a valuable symptom, being best elicited by grasping the organ between the fingers placed over the flank behind and the thumb in front. The urine, on repeated examination, will, in addition to blood, also generally reveal pus. The presence of pus indicates pyelitis or inflammation of the mucous surface of the renal pelvis or calyces; the urine is *acid*, and does not present the characters met with in bladder cases. More or less fever sometimes is associated with this pyuria, but it is not unusual to find smart rigors supervene, with a high temperature and sweating, in cases where no pus-cells are found in the urine. More or less albuminuria is usually present. Dyspeptic symptoms, and gastric disturbance amounting to severe vomiting, may occur at irregular intervals, independent of the attacks of renal pain and hæmaturia.

Diagnosis.—*Biliary colic* resembles that produced by the passing of a renal calculus down the ureter on the right side. The pain, however, does not follow the direction of the ureter, and never invades the testicle or causes tenderness or retraction of this organ, and is usually confined to the upper zone of the abdomen, often radiating to the right shoulder. There is no blood in the urine, and frequent micturition does not occur; but jaundice commonly follows, and often some distension of the gall-bladder.

Where the stone remains in the kidney the dull boring pain may be mistaken for *neuralgia*, but the marked tenderness of the kidney itself, when grasped between the fingers placed behind, and the thumb in front, is characteristic. The history of repeated attacks of pain and hæmaturia, especially after active movements, is generally sufficient to distinguish stone in the kidney from *tubercular pyelitis*; though pyuria may be present in both, blood is very seldom present in any appreciable quantity in the strumous kidney, even though micturition should be frequent.

Stone in the bladder is not likely to be mistaken for renal calculus unless some pyelitis is also present. The blood will not impart the smoky character to the urine—it will be bright red or purple, the pain will be felt in the neck of the bladder, and the urine will generally be found alkaline. The sound will settle the diagnosis of bladder-stone, and the Röntgen rays may reveal the presence of the renal calculus.

Malignant disease of the kidney leads generally to a spontaneous profuse hæmaturia, usually not aggravated by movement. The tumour in the loin is usually obvious on palpation as the emaciation and associated cachexia become established.

Hydronephrosis is eliminated by its intermittent characters and the rapidity with which the increase in the renal tumour progresses ; the history will often reveal evidence of a calculus having previously blocked the ureter during an attack of nephritic colic.

Floating kidney may be differentiated by its physical signs and previous history ; sometimes it causes an intermittent discharge of pale or blood-stained urine when torsion of the ureter has occurred. It must, however, be remembered that a stone in the kidney may in rare cases be the cause of the renal ptosis or a result of it, in which case the Röntgen rays alone will demonstrate the true condition of affairs. The nature of the stone can sometimes be guessed at by noting the intensity of the symptoms, most authorities maintaining that the uric acid calculus causes the least distress, whilst the oxalate of lime or mulberry stone usually produces a sharp localised pain which radiates downwards along the ureter and sometimes upwards towards the scapula. Phosphatic calculi in the kidney usually cause severe and constant suffering. These variations in the pain are obviously the result of the differences in the shape, smoothness, and other physical qualities of the different calculi.

The examination of the urine at regular intervals will, however, afford more reliable evidence of the chemical composition of the stone, the presence of constant acidity in the renal secretion indicating an oxalate of lime or uric acid stone, whilst a phosphatic calculus is usually accompanied by alkaline or neutral urine. The crystalline deposit, as seen under the microscope, or the naked-eye characters of any gravel or renal sand which may appear, will also aid in establishing the nature of the renal concretion.

Treatment.—Where the stone is large, and continues to cause severe pain and hæmorrhage, the rest-cure should get a trial. Sometimes *absolute* rest in bed for several weeks has been followed by cessation of all symptoms by permitting the stone to become permanently fixed in the calyces or some part of the renal pelvis.

Roberts maintained that uric acid, urates, or cystine calculi could be dissolved *in situ* by keeping the patient's urine *continuously* alkaline for long periods by the persistent administration of citrate of potash.

Nephrolithotomy is indicated when the above measures fail ; and should the kidney be found disorganised, the operation of nephrectomy, or removal of the diseased organ, must be carried out. The previous determination of the renal adequacy is necessary in order to obtain evidence of the state of the other kidney. This may be demonstrated by Kümmel's method, which is based upon the fact that, as long as one kidney is healthy, the freezing-point of the blood never falls below

– 0·56° C. The more recent method of Wright is convenient and reliable. The determination of the renal adequacy is a matter of vital importance in *prognosis*.

For the relief of the severe pain of renal colic caused by the passage of a stone down the ureter the *hot* bath is the best routine agent, the patient remaining in it, if necessary, till the first symptoms of faintness are experienced. Morphia hypodermically or chloroform-inhalation may be necessary when the agony is severe ; massage and inversion of the body rarely are of use, and often are intolerable. The effects of the hot bath may be prolonged by stupes, poulticing, and cupping.

After the passage of a stone, the agents which tend towards the prevention of further concretions should be insisted upon. These are the sparing use of animal food and the free administration of diluents in the form of rain or distilled water, and, in the case of uric calculi, the constant administration of large quantities of alkaline mineral waters.

KIDNEY, TUMOURS OF

Cystic growths have already been described on page 753. Hydronephrosis and other innocent growths are referred to under their special headings, as 'Hydatid Disease,' 'Bilharzia,' 'Pyelitis,' &c. Adenomata, fibromata, lymph-adenomata, cavernous angeiomata, papillomata, and gummata are rarely met with of such dimensions as to form palpable tumours.

Tubercle of the kidney is a common event in general tuberculosis, in which case it is a secondary affection, and rarely gives rise to any definite symptoms, the disseminated grey or yellow tubercles interfering little with the function of the organ as they lie scattered through the cortical or medullary areas.

Primary tubercle of the kidney shows itself as a pyelitis or pyonephrosis, constituting the condition known as strumous pyelitis, and will be found described under the heading 'Pyelitis.'

MALIGNANT DISEASE OF THE KIDNEY

This occurs in the forms of sarcoma and carcinoma, which may be either primary or secondary. The sarcomatous form is generally primary, whilst in carcinoma, as in tubercular disease, the malignant nodules may be deposited throughout both kidneys during the progress of a malignant growth which has invaded other abdominal or pelvic organs, the implication being unattended by any special renal symptoms or signs. Primary carcinoma of the kidney also occurs.

Sarcoma of the kidney is much more common than carcinoma, and it often occurs during infancy or early childhood,

usually before the age of five. It is nearly twice as frequently seen in boys as in girls, and the proportion of males affected when the disease occurs after middle-life is also much greater. The growth in early life is sometimes bilateral, consists of round or spindle cells, occasionally with striated muscular fibre, and soon destroys all traces of the renal secreting tissue, assuming, in a period of some months, colossal proportions, so that a considerable area of the abdomen may be occupied by the tumour or tumours. The growth does not destroy the renal outline, even when the secreting structure has already disappeared ; and when large, soft projecting masses may be felt on the surface of the tumour. There is little or no tenderness and pain present, and hæmaturia is rare ; secondary growths are common in the lymphatic glands, the liver, and lungs. Emaciation becomes extreme, and the abdominal veins prominent ; the disease proves fatal in from six to twelve months in the great majority of cases.

Sarcomatous tumour of the kidney in the adult, though it soon assumes large proportions, is not so rapidly fatal as in the child. It is almost invariably confined to one organ, and the structure is seen to be made up of spindle-cells, with much striated muscular tissue. Hæmaturia is often present and usually in large amount, often with considerable pain, and a sense of weight or dragging in the loin on the affected side. Emaciation may be absent for a long period, but finally it becomes marked, and secondary deposits appear in the lymphatic glands and other organs.

Many of the so-called renal sarcomatous tumours originate in the adrenal tissue in adults, and occasionally in the accessory adrenals, in which case hæmaturia does not occur till a late stage, or never appears at all. These growths (known as *Grawitz's tumours*) consist of hypertrophied adrenal tissue, and can often be successfully removed by surgical methods.

Carcinoma of the kidney, when primary, is comparatively rare, and, like sarcoma, is much more common in males. Occasionally there will be a history of trauma ; the growth is nearly always of the encephaloid type, and unilateral. It does not rapidly tend to produce extreme emaciation or secondary deposits, running generally a very much slower course than carcinoma of the liver does. Eventually great wasting and secondary tumours in other organs and tissues supervene. The tumour, in the slowly progressive cases, may reach large dimensions ; it is uneven in its outline, and at different parts affords evidence of different degrees of consistency, being in some places almost fluid in the larger bosses from colloid changes, whilst in others the nodules may feel tense and hard from hæmorrhage, fibrous trabeculæ, or adhesions. Tenderness is usually present, and pain is rarely absent ; this latter may be of a severe neuralgic

character, radiating downwards along the inside of the thigh, and associated with a distressing dragging sensation of weight in the loin. Owing to the presence of adhesions, the tumour is but slightly movable and, when large, is little, if at all, influenced by respiration ; it may bulge backwards and be visible and palpable from behind ; the colon will be always in front of it. On the left side, the stomach and spleen will be displaced inwards, and on the right the liver will be found elevated.

Hæmaturia is usually profuse, and often blocking of the ureter and urethra occurs from the large clots which form portions of a cast of the renal pelvis or ureter ; rarely fragments of cancerous tissue or cells of the characteristic type may be found in the urine.

As the disease progresses, the growth, which begins in the cortex and is encapsuled at first, invades other organs and tissues in the vicinity, and finally may involve the spinal cord, vena cava, or aorta, or the secondary deposits may interfere with the functions of vital parts at a distance from the kidney, as may sometimes be seen in the interference with the brain-centres. Œdema of the lower extremities or ascites may become a prominent pressure-symptom.

Diagnosis.—From *benign growths*, which are rare, sarcoma and carcinoma are distinguished by their larger dimensions, by the progressive weakness and emaciation which they produce, by the hæmaturia, which usually is independent of exercise or jolting movements, and, in the case of cancer, by the severity of the pain.

Hydronephrosis is excluded by its history of old calculi and the intermittent discharge of large quantities of clear urine, and by the uniformly soft and fluctuating character of the tumour. The hydronephrosis occurring in *movable* kidney is also differentiated by the same characters, aided by the history of the case and the evidence of ptosis of other organs.

Hydatid disease has a long history, in which emaciation, pain, and hæmorrhage are usually absent. The tumour is smooth, tense, and fluctuating ; its contents on aspiration are characteristic, and sometimes hooklets or small cysts are found in the urine after attacks of masked renal colic.

Cystic disease of the kidney is of very long duration. Both organs are usually involved, and the congeries of cysts may be palpated on the surface of the organ ; but it may require the use of the aspirating needle to settle the diagnosis in those cases where cystic disease has progressed to the extent of inducing constitutional symptoms before being discovered. The signs of a chronic interstitial nephritis, as seen in the characters of the urine and vascular system, will be strongly in favour of cystic disease. The urine in malignant disease

of the kidney very seldom contains albumin, unless some blood be present.

Bilharzia affecting the kidney may cause a difficulty in the diagnosis in Egypt and other parts of Africa, where the disease is common; but even when the main signs of the disease are confined to the pelvis of the kidney, constituting a pyonephrosis, there will be evidence of bladder-trouble, and the ova may be found in the urine. The hæmaturia of this disorder presents distinct vesical characters.

Pyonephrosis will present in its history and symptoms evidence of the cause of the pyelitis which has produced it; thus a history of calculi or of cystitis will often be obtainable. When the tumour discharges its contents by the ureter, the intermittent pyuria and synchronous diminution in the bulk of the tumour are characteristic, as are also the frequent rigors and pyrexia. The pyelitis and pyonephrosis of tubercle rarely lead to any considerable degree of hæmaturia; there is evidence of tubercle elsewhere, often in the testicle, and bacilli may be found in the urine.

Stone in the kidney, though associated with pain and hæmaturia, seldom eventuates in the production of a palpable tumour unless pyonephrosis or abscess results; and the hæmaturia of renal calculus can usually be traced to active movements or joltings, whilst in sarcoma and cancer the bleeding is spontaneous.

Disease of other organs may be mistaken for renal cancer or sarcoma, thus:

Splenic enlargement is recognised by the outline of its distinct edge, with the characteristic notch, and by the smooth uniformly firm feel of the body of the tumour, which has the colon behind it; moreover, its ascent and descent in respiration are much more obvious than in renal tumours.

Hepatic disease causing enlargement may be mapped out by careful percussion, the lower edge being felt as the tumour moves freely up and down during respiration, and the urine will contain neither blood nor pus. In carcinoma or other tumour of the kidney there will be a band of resonance between its upper margin and the edge of the liver, unless the tumour be very large. The position of the colon in front of the enlarged kidney is a most important feature in the diagnosis of all renal tumours. When the renal tumour is so large as to cause elevation of the liver, the anterior sharp edge of this organ may often be felt tilted forwards in contact with the abdominal wall.

Sarcomatous growths originating behind the peritoneum are liable to be mistaken for renal tumours; they occur in children, and come forwards towards the middle line, pushing the kidney out of its place. The rare *fibrolipomata* of retro-peritoneal origin are most frequently met with in women

about middle-age, though a few cases have been reported in children ; they are painless, and cause no symptoms even when of colossal size.

Adrenal growths, as already stated, never cause hæmaturia, unless in the rare cases of sarcomatous nature, when the kidney becomes involved in the later stage ; they are solid, and pursue a slow course ; they are liable to be mistaken for the renal sarcoma of infants, but they are never bilateral.

Cancer originating in a floating kidney may cause most puzzling signs. In one case under the writer's observation the growth became enormous ; but the floating tumour was known to have existed for many years before, and there was no difficulty in the diagnosis, which was verified on *post-mortem* examination.

Prognosis.—This is always grave and practically hopeless, unless when an early diagnosis has led to the removal of the diseased organ when comparatively small. It must be remembered that sarcoma of the kidney in the adult may pursue a very slow course, even when hæmaturia is copious.

Treatment.—The early removal of sarcomata affords the only hope of prolonging life, and good results may be expected from extirpation of adrenal growths ; but infantile sarcomata may usually be regarded as beyond operative treatment, secondary deposits being the rule.

Carcinoma of the kidney has in a few cases been operated on with success, but, as a rule, by the time the diagnosis has been rendered certain the operative stage has passed.

LARDACEOUS DISEASE

This condition, also known as Amyloid and Waxy Degeneration, is the result of the deposition of an amyloid substance (which is an albuminous body in coagulated form) in the small arteries and capillaries of the kidney, spleen, liver, suprarenal bodies, and intestinal mucous membrane. At a later stage the connective tissue may become infiltrated, but the most remarkable feature of the degenerative process is that it always commences in the small vessels. In the spleen, the entire parenchyma becomes transformed into amyloid substance in the so-called ' diffuse ' degeneration ; whilst in the ' sago spleen ' the infiltration is limited to the Malpighian follicles and adenoid sheaths.

The nature, origin, and exact composition of the amyloid substance are still subjects requiring further research ; there is, however, evidence to show that lardacein is closely allied to ordinary egg-albumin, and that it differs in its composition somewhat according to the nature of the organ in which it is deposited.

Under 'Amyloid Disease of the Kidney' (page 129), the naked-eye characters, and the chemical reaction of the amyloid body, the pathological anatomy, and etiology are dealt with. The causes in general are the same, whatever organ has first become the seat of the disease. The condition is always secondary to some other affection, the most common being tuberculosis of the lung, especially where large suppurative cavities have been established, or the suppuration following tubercular caries of the spine or joints; but the extensive production of pus from any cause, as from the large cutaneous areas destroyed by burns, from empyemata, bronchiectasis, the ulcers of dysentery, and other conditions, may induce amyloid. Syphilis is a powerful factor, even when no necrotic processes are at work. When amyloid disease supervenes upon any of the forms of Bright's disease, probably syphilis or phthisis has been previously operating in producing a general dyscrasia.

The advent of the degeneration may be as early as three or four months after the establishment of extensive suppuration; more frequently it is much later, and its duration may be indefinite.

The disease is never confined to a single organ; the spleen and kidneys are almost invariably affected in every case, and, in the great majority of cases, the liver, but to a less extent the intestinal mucous membrane.

Amyloid disease of the intestine occurs in the majority of long-standing cases in which the spleen and kidneys are involved. It is never confined to the intestine, and usually only appears at a late stage—a considerable period after the spleen and kidney have become affected.

The symptomatology is of secondary importance, owing to the facts just mentioned, as almost invariably there will be previous evidence of the disease in the spleen and kidney, and probably also in the liver.

Diarrhœa is usually the first, and often the only, evidence of the invasion of the intestinal mucous surface. Occasionally blood may appear in the motions, indicative of the formation of ulcers in the lower part of the small intestine, which is the part most commonly involved.

Where tubercular disease in the lung or in bone is supposed to have produced lardaceous degeneration in the intestinal mucous membrane, it becomes a very difficult point to eliminate *tubercular ulceration* of the bowel, even should the spleen and kidney be obviously affected. The presence of masses of adherent glands and other signs of adhesive inflammation of the peritoneal membrane, with the alternation of diarrhœa and constipation, along with much blood in the loose stools, will settle the case in favour of tubercle.

The prognosis will be always grave because usually the intestinal surface is involved late, except possibly in some syphilitic cases, which may yield to iodides in full doses.

The treatment will necessarily be confined to the administration of astringents, combined with enough morphia to inhibit the exalted peristalsis.

Lardaceous disease of the spleen and liver will be referred to under 'Spleen' and under 'Liver Diseases.'

LARYNGISMUS STRIDULUS

This is also known as Child-crowing. It is a spasmodic affection of the larynx, occurring nearly always in young children who are suffering from rickets.

Etiology.—The disease is usually limited to children between the ages of six months and two or three years; very occasionally it appears in children up to seven or eight years old. Rickets almost always is found to be the primary cause. Tetany, when not of rachitic origin, in some cases is associated with the laryngeal spasm. Malnutrition, congenital hydrocephalus, and a microcephalic state are etiological factors in some non-rachitic children.

The exciting cause of an attack in a predisposed child is often seen to be a fit of temper, or excitement, or a paroxysm of whooping in pertussis or measles, the eruption of a tooth through an inflamed gum, or the trickling of milk into the larynx in rapid or greedy attempts at swallowing or sucking, especially in the presence of congenital adenoid growths. More frequently, however, no such exciting cause is obvious. It is very doubtful if enlargement of the thymus gland acts as a predisposing or an exciting cause, but in older children enlarged bronchial glands may cause attacks up to the tenth year.

Symptoms.—There is never any indication of an inflammatory condition of the larynx, or hoarseness, such as is usually present in spasmodic croup. The seizure begins suddenly with a noisy or stridulous inspiration, caused by adductor-spasm, which rapidly becomes so complete as entirely to arrest respiration. The child struggles violently for breath, with its head retracted and the neck prominently arched forwards, the face becoming livid; or a death-like pallor, with perspiration, appears. Twitching of the facial muscles, with carpo-pedal contractions, are common in severe cases, and these may rapidly pass into a general convulsion, during which deep cyanosis occurs. The spasm, which may last for a half to two minutes, relaxes as suddenly as it commenced, and the air is drawn into the lungs with a crowing sound,

after which the lividity disappears and the child speedily recovers.

Death may occur in the first attack, and if the seizure has not been observed, the diagnosis remains obscure. More frequently the attacks are of a milder nature, the child seems voluntarily to hold its breath for fifteen to thirty seconds whilst awaking from sleep; no spasms of the face, hands, or feet are noticeable, and little or no stridor may be heard. Such attacks may frequently recur during the day and sometimes in the night, and are liable to be induced by crying or fits of temper.

In the adult, laryngeal spasm may supervene upon any structural disease of the larynx, or upon irritation of the recurrent laryngeal nerves, or it may be of centric origin.

Diagnosis.—The presence of rickets, the abruptness of the onset, the rigid fixation of the chest, the transient nature of the attack without hoarseness, aphonia, or cough, are characteristic. In doubtful cases, the strong flexure of the thumbs, which are concealed in the palms by the contracted fingers, and the inturned flexed feet, will demonstrate the nature of the case when no stridor is present.

Prognosis.—This is always serious, as death may occur in any case without warning, though the great majority of cases terminate favourably. Semon and Williams regard the prognosis in those cases where there is no stridor as especially grave.

Treatment.—During the attack, the best procedure is to dash cold water on the child's face, which nearly always cuts short the seizure. Between the attacks, when these are frequent, cold sponging of the thorax may be resorted to whilst the child is partially immersed in a hot bath. When the attack is prolonged, the tongue should be forcibly pulled forwards; tracheotomy may be demanded, followed by artificial respiration. Chloroform-inhalation may be resorted to in recurring severe attacks. The state of the gums and bowels should be seen to, and the recognised treatment for rickets persisted in.

LARYNGITIS, ACUTE

This appears as an acute catarrhal inflammation of the mucous membrane of the larynx, often associated with a similar condition in the trachea and larger bronchi.

Etiology.—Exposure to cold, especially after the inhalation of the air in rooms over-heated by gas and vitiated by over-crowding and imperfect ventilation, is the most common cause. The inhalation of irritating gases, as chlorine or ammonia, and the scalding of the epiglottis produced by hot liquids and steam, or the application of caustics, are sufficient

to excite severe and even fatal laryngitis. Microbic agents, such as those which produce epidemic catarrh, grippe, and measles, usually affect the larynx, as well as the nasal and bronchial surfaces. The prolonged use of the voice in loud speaking is a common cause, and this is often intensified by the sudden exposure to the cold air which so usually follows public speaking and singing. The attack in rare cases may be caused by the uric-acid diathesis, though this is more liable to act as a predisposing influence.

Symptoms.—The first symptom usually is hoarseness, which may deepen in severe cases into complete aphonia, especially in women. With the huskiness of the voice there is a sensation of tickling, dryness, and soreness, which causes a painful dry cough, intensified by inspiring cold or hot air. Phonation is more or less painful, and loud speaking impossible ; even inspiration sometimes causes pain, and the act of swallowing is not unusually accompanied by some soreness.

In young children, a slight degree of tumefaction of the mucous membrane gives rise to the severe symptoms recognised as spasmodic croup (page 254). In adults there is rarely any dyspnœa, unless the cause of the laryngitis be such as to produce œdema. A considerable amount of relief is usually experienced as soon as expectoration begins to accompany the act of coughing, and often the tenderness of the larynx, which is commonly present on palpation, disappears. The mucus which is coughed up may be tinged or streaked with blood. Constitutional symptoms are mild or absent, unless when the catarrh of the laryngeal membrane is a part of the general infection of influenza, measles, or epidemic catarrh, in which case there may be abundant muco-purulent secretion and tracheal soreness, with fever and malaise. The laryngeal mirror will reveal swelling and redness of the vocal cords and mucous membrane, with the presence of more or less mucoid secretion, and occasionally some abrasions. The cords may be seen to move less freely, owing to their tumefaction, and in severe cases ecchymosis or streaks of blood may be visible.

In mild cases, the attack passes off in a few days, but it may remain for weeks, especially in gouty and influenzal cases, and in hysterical subjects, or in neurotic patients with menstrual irregularities.

Where the laryngeal inflammation has been caused by the inhalation of superheated steam or irritating gases, or by the application of corrosive substances, the symptoms may be those of intense dyspnœa and cyanosis, which may threaten suffocation by œdema or spasm.

Diagnosis.—In children, *laryngismus* is easily eliminated by the characteristic symptoms mentioned upon a previous

page, especially by the absence of hoarseness. *Diphtheritic laryngitis* is recognised by the presence of false membrane, which is usually also visible on the fauces, and in doubtful cases by the recognition of the Klebs-Loeffler bacillus in the secretions obtained by swabbing. Usually the laryngeal examination settles the diagnosis of catarrhal laryngitis in the adult without difficulty, and easily eliminates *hysterical aphonia*.

Prognosis.—In catarrhal laryngitis the prognosis is always good in adults, the only exception being in those rare cases caused by corrosives. The influenzal cases, though never fatal, may be tedious in their course, and may leave a considerable degree of weakness of the voice behind, which is slow in disappearing. The prognosis in spasmodic croup is always good.

Treatment.—Rest to the voice is most important, in a room with a moist warm temperature (65° F.). Where the local symptoms are unusually severe, warm applications to the larynx, and the steam from very hot water in a large basin, may be freely inhaled, a few drachms of friar's balsam or hemlock-juice being added to the hot fluid. Carbolic spray (1 in 100) is also soothing and checks cough. A diaphoretic mixture containing ipecac. and tartarated antimony, in doses bordering upon the nauseating, may be given every three or four hours. In children, the symptoms of laryngeal irritation should receive the promptest attention, and the speedy administration of an antimonial or ipecac. emetic will prevent an attack of spasmodic croup.

LARYNGITIS, CHRONIC

Etiology.—The most common cause of chronic catarrhal inflammation of the larynx is undue exercise or over-use of the vocal organs, especially in those who speak in the open air, as street-preachers, or those whose avocations involve the shouting of street-cries. Any of the causes which produce acute attacks, if long-continued, may induce the chronic form, as the breathing of air charged with irritating dust or corrosive gases. It is a noteworthy fact that chronic laryngitis is very rarely seen amongst the operatives of flax-mills, though they generally suffer from the irritation of the flax-dust in the lower part of the respiratory tract; whilst stone-dressers, on the other hand, often suffer from chronic catarrh of the laryngeal membrane.

The condition is very commonly associated with the special forms of chronic laryngeal disease produced by syphilis, tubercle, and cancer; in rare cases it may be an extension of a very chronic variety of pharyngitis; more commonly it is

found associated with atrophic rhinitis. It is very doubtful if the use of tobacco ever leads to chronic laryngitis, though it is often regarded as an etiological factor.

Symptoms.—The main feature is the marked hoarseness of the voice usually associated with a sensation of tickling, and an habitual dry cough. When the voice is exercised for any length of time, aphonia may suddenly supervene in the middle of a sentence or word ; it may be constantly present in severe examples. The act of speaking, though not painful, is usually accompanied by marked fatigue ; after a few moments' use of the larynx the hoarseness or aphonia is relieved till fatigue sets in.

In the *dry* form associated with atrophic rhinitis dry pellets or crusts may be coughed up with difficulty ; in the ordinary catarrhal form a little toughened mucus is usually expectorated.

The mirror will reveal some congestion of the cords, which are often thickened or somewhat nodulated in an irregular manner, and do not approach each other on phonation as in health, owing to loss of muscular tone. One cord is usually more affected than the other, and sometimes the affection seems unilateral. The ventricular bands are sometimes much thickened, and the inter-arytenoid fold swollen. In a variety of chronic laryngitis styled the *sub-glottic* there is much thickening and hypertrophy of the sub-mucous tissue below the cords which considerably narrows the lumen of the larynx. In the *glandular* variety the induration or thickening is chiefly the result of swelling of the racemose glands.

Mucus may be seen on the vocal cords, arytenoid commissure, and ventricular bands. In the dry form crusts may be observed ; ulcers are rarely visible, though slight superficial abrasions or shallow erosions may occasionally be seen.

Diagnosis.—The laryngeal examination usually is sufficient to demonstrate the case when taken together with the clinical history. The greatest difficulty arises in the exclusion of *tubercular disease* in its early stage ; after a time the pallor of the membrane and the localisation of the tubercular infiltration to the epiglottis and arytenoid cartilages are characteristic. The state of the lungs and the presence of the tubercle bacillus must in all cases be carefully investigated.

A unilateral swelling or infiltration of one cord, producing marked loss of motion in it, when occurring at the cancerous period of life, should arouse suspicion of *malignant laryngitis*.

Prognosis.—This will depend upon the cause and the duration of the disease, and is usually good in all cases when the patient can give himself up to a prolonged rest of the voice by a temporary or permanent change of avocation,

though a tedious course of convalescence may be safely predicted.

Treatment.—Prolonged voice-rest is essential, and the removal of every predisposing or exciting cause, as exposure to cold and variations of temperature. Often a change of climate is necessary for a time if the patient is to hope to resume his avocation as a public speaker or singer.

Where acute symptoms have supervened upon the chronic affection, the soothing sprays and inhalations indicated in acute laryngitis should be resorted to for a short time. The most reliable of all methods is the patient application of astringent solutions by means of the brush and the laryngeal mirror. (A list of these local remedies, with their appropriate strengths, will be found detailed in the last edition of the author's 'Dictionary of Treatment,' upon pages 500 to 506.) The solution applicable to the majority of cases is that of chloride of zinc (1 in 20), which should be applied daily. In very chronic cases 1 in 8 of nitrate of silver may be freely used. After a few weeks the patient may begin to treat himself by the use of a weak chloride of zinc spray (1 in 200).

LARYNGITIS, MEMBRANOUS

All cases of inflammation of the larynx in which a false membrane is formed are now regarded as of the nature of true diphtheria. Under the pathology of 'Diphtheria of the Larynx' on page 304, the subject is discussed, and facts are stated, as observed by the writer, which have convinced him of the existence of a form of membranous laryngitis which is not produced by the Klebs-Loeffler bacillus, though the clinical symptoms are identical save that paralytic sequelæ do not occur and the disease is not communicated to others.

LARYNGITIS, ŒDEMATOUS

Œdema of the epiglottis, aryteno-epiglottic folds, ventricular bands, and the sub-mucous tissue below the cords may occur without any true inflammatory action as part of the general dropsy caused by chronic valvular disease of the heart and Bright's disease, or it may be the result of a local dropsy or anasarca, produced by the pressure of tumours in the neck or mediastinum. Such cases do not strictly fall under the title 'Œdematous *laryngitis*'; but usually for clinical convenience all forms of swelling of the mucous or sub-mucous tissues in this region are usually considered as belonging to the heterogeneous group of affections styled œdema of the glottis, œdema of the larynx, or œdematous laryngitis.

Etiology.—Trauma supplies the most typical examples of the disease, as is witnessed in the severe inflammatory swelling which rapidly follows the swallowing of scalding liquids, as boiling water or tea, ammoniacal and corrosive liquids, or the entrance into the larynx of some jagged body, as a piece of splintered bone or the pointed fixing of an artificial tooth. Any cause which produces a severe acute catarrhal laryngitis, as exposure to cold, especially in the subjects of chronic renal affections, may induce formidable œdema.

Tubercular, syphilitic, and malignant diseases, and any condition which induces necrosis of the cartilages, may eventuate in œdematous laryngitis. Severe inflammations the result of microbic infection, as erysipelas, diphtheria, cellulitis, and tonsillitis, and other phlegmonous conditions of the neck, mediastinum, or fauces, and the microbes which produce scarlatina, measles, typhus or typhoid fevers, small-pox, hydrophobia, and influenza, may be the cause of formidable œdema.

There is also met with a rare form of the disease described as angio-neurotic œdema, probably the result of a poison like that which produces urticaria, in which, without any evidence of inflammatory action, severe swelling of the laryngeal tissues may occur in adults ; as already mentioned, the condition may be secondary to general or local dropsy. Iodide of potassium or sodium has been known to produce a fatal œdema of the larynx, probably by the irritation which the drug produces on the vaso-dilator fibres on its elimination by the laryngeal mucous membrane below the level of the vocal cords.

Symptoms.—These are acute and formidable, commencing suddenly with painful deglutition, hoarseness rapidly passing into aphonia and dyspnœa (which is usually greater on inspiration), and marked stridor.

The laryngeal mirror will reveal great swelling of the epiglottis and of the aryteno-epiglottic folds. In the inflammatory cases the tumefaction is of a bright red, but when the œdema is passive the swelling is pale in colour. The vocal cords are but slightly affected ; the œdema may be confined to the larynx below their level, the swelling being visible between them.

Diagnosis.—This can often be made out by digital examination, the swollen folds of the aryteno-epiglottic tissue being easily felt and the epiglottis rendered visible on depression of the tongue.

Prognosis.—The forecast is always grave. In the cases produced by scalding water, steam, or ammonia death may occur within a period of minutes. In the phlegmonous cases the outlook is but slightly less serious ; but where the œdema is passive, as in heart and kidney disease, the prognosis is

usually favourable unless there be evidence of glottic spasm.

Treatment.—This should be prompt. In rapidly progressive cases no time should be lost in watching the result of ice to the larynx or leeches to the front of the neck, but scarification of the epiglottis and of the aryteno-epiglottic folds should be performed by means of a curved bistoury guarded to within a quarter of an inch of its point. If speedy improvement be not obvious, tracheotomy should be performed without delay ; intubation is difficult, and may be impossible. Pilocarpine hypodermically, though highly recommended, is dangerous, owing to its liability to produce dropsy of the lung unless the patient be enveloped in a hot pack before its administration. The danger of spasm of the glottis may be minimised by the rectal injection of 100 grs. or more of bromide of soda in 3 oz. water.

LARYNGITIS, SPASMODIC

This is the name often wrongly applied to laryngismus stridulus, in which, however, there is no element of inflammation. The term, if used, should be restricted to the condition known as the false or spasmodic croup of childhood, in which the element of spasm of the laryngeal muscles supervenes upon a slight catarrhal laryngitis, as described under 'Croup,' page 254.

Spasm of the glottis in adults may be the result of some centric disturbance, as in hysteria, epilepsy, hydrophobia, tetanus or chorea, and the laryngeal crises of locomotor ataxia. More frequently it supervenes, as already mentioned, upon any inflammatory state of the larynx. It may be produced by the irritation of the recurrent laryngeal nerve, occasioned by the pressure of aneurismal or mediastinal growths, or by the introduction of foreign bodies into the larynx.

When the spasm in these cases threatens suffocation, if it does not yield to chloroform-inhalation the operation of tracheotomy should be performed without delay, the subsequent treatment being that of the primary cause of the spasm, as already detailed.

There is a form of adductor-spasm known as Phonic spasm or Spastic aphonia, which only occurs on attempts at speaking or singing. The affection is allied on the one hand to the barking cough of puberty, and on the other to the neuroses of which habit-spasm or writer's cramp is the type. It is most frequently met with in public speakers or singers, and is usually invulnerable to everything but change of occupation or prolonged rest.

LARYNGITIS, SYPHILITIC

Syphilis of the larynx may be of hereditary origin: more frequently it is a late secondary or tertiary manifestation of the acquired disease ; but the affection may appear at any stage of the primary disorder, from the appearance of the cutaneous rash till the expiration of more than a quarter of a century after the original infection. In one instance the writer witnessed its advent thirty-five years after the primary sore.

Symptoms.—During the eruption stage a catarrhal laryngitis is often observed apparently identical in its symptoms and laryngeal signs with ordinary laryngeal catarrh, with its hoarseness of voice and tickling cough, and only to be diagnosed by its indefinite duration and liability to return in spite of ordinary antisyphilitic remedies. Usually there are none of the characteristic manifestations seen on the other mucous surfaces, as condylomata, but shallow abrasions or superficial ulcers are occasionally observable on the upper surface of the epiglottis or aryteno-epiglottic folds or cords.

The tertiary forms of the disease exhibit the usual polymorphous characteristics of late syphilis. A diffuse small-celled infiltration may invade the entire tissues of the larynx, causing at first thickening, which may afterwards eventuate in considerable deformity as the resulting fibroid tissue shrinks, or the new cell-growth may break down in parts and form deep ulcers. More commonly small gummata form, usually about the base of the epiglottis or its folds, in the sub-mucous tissue ; these soften and break down, leaving deep ulcerations which may be visible also on the folds or inferior portions of the larynx. The healing of these ulcers, owing to the contraction of the fibroid tissue, produces stenosis and characteristic deformities which seriously interfere with respiration, phonation, and even deglutition. The extension of the ulceration often involves the cartilages as it eats its way outwards, perichondritis preceding the necrosis, which latter process may be so complete as to destroy the entire epiglottis, and arytenoids and a portion of the cricoid and thyroid cartilages.

The symptoms vary with the exact situation of the destructive lesion. Hoarseness sometimes amounting to complete aphonia exists, and dyspnœa may be a marked feature. Pain and difficulty in swallowing are sometimes present, but as a rule there is little pain in the course of syphilitic laryngitis—a feature of importance as contrasting with malignant disease.

Cough is generally present, but not usually severe or painful. It may be associated with hæmorrhage, spasm, or the expectoration of pieces of dead cartilage.

Œdema of the glottis, laryngeal spasm, hæmorrhage, or paralysis of the larynx may suddenly supervene in any severe case.

Diagnosis.—The main point in the diagnosis of the laryngeal catarrh which occurs in the active secondary stage of syphilis is the recognition of the other unmistakable signs of the primary disease. In the tertiary syphilitic forms of laryngitis, the presence of other tertiary syphilitic lesions, as nodes, paralysis of eye-muscles, hepatic gummata, ataxia, arterial degeneration, &c., and, in the absence of these, the history of a remote infection, with its secondary eruption, mucous patches, and iritis, is valuable evidence, but the laryngoscopic examination should always be the deciding factor in every case. The deep ulcerations, with their sharply defined undermined margins and their central dead or inactive zone, are characteristic, as are also their old stellate scars and puckerings.

Cancer may be eliminated easily in its late stages by the cachexia and secondary glandular enlargement in the neck; but in the early stage the diagnosis must often remain doubtful for a long period, and only becomes certain after the histological examination of a fragment of the detached growth and the futility of large doses of iodides.

Tubercular laryngitis is differentiated by the age of the patient, the pallor of the laryngeal membrane, and the thickening of the epiglottis and involvement of the arytenoid cartilages, and the superficial nature of its worm-eaten ulcers. The discovery of the tubercle bacillus and the presence of pulmonary or abdominal tuberculosis are also conclusive.

Prognosis.—This is not dangerous to life unless where cicatricial deformity and loss of substance interferes with swallowing and breathing. The disease, when recognised early, yields to vigorous treatment continued for long periods.

Treatment.—In the early stages mercury should be pushed by the inunction method, and even in late tertiary cases this plan should be persevered in. Iodides alone often produce striking results, but as a general rule it is wise to commence with mercury, or to resort to short courses of the metal in the intervals when the iodide treatment is suspended. Iodide of sodium must be given in large quantities, beginning with doses of 10 to 15 grs. thrice daily, which are rapidly increased till 3 or 4 drs. are administered during the twenty-four hours.

When cicatricial deformity has occurred, tracheotomy and the permanent wearing of a tube may be necessary for the remainder of life.

Dilatation of the stenosed opening has been successfully accomplished by bougies; cicatricial webs have been divided by cutting instruments and the electric cautery; and in some cases a portion of the deformed larynx has been successfully removed by the knife.

LARYNGITIS, TUBERCULAR

As its name implies, this is an inflammation of the laryngeal tissues produced by irritation caused by the tubercle bacillus.

Etiology.—The disease is almost invariably secondary to pulmonary phthisis, and in practice may safely be regarded as never of primary origin, though a few such cases have been recorded. Young adult males are affected more frequently than females. About one in every four phthisical patients presents some signs of the disease when carefully examined, though the laryngeal symptoms and signs may be obvious when the lung-disease is in an incipient stage.

Pathological Anatomy.—This corresponds to the laryngeal picture seen during life by the aid of the mirror. There is infiltration of the mucous and sub-mucous tissues, especially in the regions of the epiglottis, the arytenoid cartilages, and aryteno-epiglottidean folds. At a later stage ulceration is found, and the ulcers are characteristic ; they usually show broad bases and are seldom as deep as those witnessed in syphilitic disease, though occasionally they may penetrate to the perichondrium of the arytenoid cartilages, which may become necrosed. The epiglottis and true vocal cords may suffer in the general ulcerative process. The affected membrane is usually marked by extreme pallor, and occasionally small masses of coalesced caseous tubercles may be seen in the stage of breaking down, surrounded by a pale œdematous tumefaction of the mucosa. Under the microscope the tubercle bacilli may be found in the small greyish tubercles studded throughout the membrane, being in the earlier stages more numerous in the vicinity of the blood-vessels.

Symptoms.—The huskiness or hoarseness of the voice characteristic of chronic laryngitis is usually the first indication of the involvement of the larynx, and this is accompanied or preceded by a troublesome irritating cough, which can usually, though not always, be distinguished from the cough caused by the pulmonary mischief ; it is often painful. Dysphagia is an early symptom in some cases, and usually a persistent and distressing complication at a later stage, when extensive ulceration has involved the epiglottis so that fluids find their way into the larynx, producing severe paroxysms of painful cough and distressing dyspnœa upon every attempt at swallowing. From ulceration, or paralysis of the cords caused by the pressure of enlarged glands the voice may be completely lost. Not only may cough and swallowing produce severe pain, but this may be constantly present in the intervals, though to a less extent.

The laryngeal mirror will reveal the appearances described in the paragraph on the pathological anatomy of the affection.

The most important of these is the extreme pallor of the mucosa which is characteristic of tubercular disease of the larynx in all its stages. The infiltration and dense pale œdema of the epiglottis and of the region in the immediate vicinity of the arytenoid cartilages, even before the formation of the numerous bilateral shallow ulcers, are usually sufficient to establish the diagnosis. At a later stage the extensive destruction of the epiglottis and the infiltration and ulceration of the cords are equally characteristic. The arytenoid cartilages may be expectorated in their entirety ; more commonly they are shed in necrosed fragments.

Diagnosis.—The laryngeal signs, as already stated, are seldom likely to be mistaken, and the diagnosis is rendered certain by the discovery of tubercular lung-affection and the demonstration of bacilli in fragments of expectorated tissue and in the muco-purulent discharge.

Cancer is often unilateral. The new-growths are generally larger than those produced by caseating tubercular nodules ; there is marked congestion in their vicinity ; hæmorrhages are not infrequent ; and there is commonly much increase of salivary secretion and fetid tenacious discharge, and, at a late stage, enlargement of the cervical glands.

Syphilitic laryngitis is differentiated by the characters already described upon page 780, chief amongst which is the rapidity and depth of the ulcerative process and the absence of general pallor. The ulceration may be confined to one side of the larynx, or it may extend rapidly in any direction, but it never exhibits the numerous small shallow ulcerating spots distributed over both sides of the larynx. Other evidence of syphilis is usually obtainable.

Lupus of the larynx is almost always secondary to that of the skin of the face after the disease has attacked the nose and pharynx. It is extremely slow in its progress, is usually unaccompanied by pain, and is found to be cicatrising at one part whilst eroding the tissues in the opposite direction.

Prognosis.—This is as a rule decidedly unfavourable, the degree of seriousness being usually in proportion to the activity and extent of the pulmonary lesion. When the disease in the larynx is first seen in its early stage, and the phthisis is confined to one apex and the family history is good, a hopeful prognosis is often possible, the forecast being influenced by the same considerations which influence the prognosis in pulmonary tuberculosis and scrofula.

Treatment.—In the earlier stage of the disease, when the signs and symptoms are those of a chronic catarrhal laryngitis, the remedies are those indicated in phthisis, in addition to soothing sprays, as 1 in 100 carbolic acid, and weak astringent applications. When ulceration has occurred, much may be done to relieve cough, pain, dysphagia, and dyspnœa by fine

sprays of menthol dissolved in liquid paraffin or oil (1 in 10), or by the direct swabbing of the ulcerated surface by a solution of twice this strength, which destroys sensibility as well as proves lethal to the bacilli. After its use, swallowing, which was before painful or almost impossible, may become comparatively easy. Cocaine and iodoform applications also afford temporary relief. When a more radical attempt is justifiable, strong lactic acid may be applied directly to the ulcerated spots by the surgeon, after curetting. The electric cautery has been also successfully employed. The result of these surgical methods has been unquestionably to cure the condition in a not insignificant percentage of cases, or to make tracheotomy unnecessary and to prolong life in comparative comfort.

The difficulty of swallowing may be lessened by the administration of food in the form of a thick liquid and by training the patient to swallow whilst his head hangs over the side of a couch or bed. It is needless to say that every agent proved useful in the treatment of phthisis should be persevered with, as creosote, cod-liver oil, and change to a suitable climate in which the waking hours may be spent in bright sunshine.

LARYNX, CANCER OF

Malignant disease affects the vocal apparatus more frequently in males than in females, and the writer, judging from his own limited experience, is led to believe that laryngeal cancer occurs usually at a later age than malignant disease in other organs. The disease is usually primary, and of the epithelial type, sarcoma being rarely met with.

The vocal cords are more frequently the first parts to be affected, one always suffering before the other ; but no portion of the larynx is exempt. When the disease commences outside the larynx proper, as proved by Semon's figures, the posterior surface of the cricoid is the most frequent site.

Symptoms.—These are hoarseness sometimes ending in aphonia, cough and expectoration, or hawking up of saliva mixed with bloody and fetid discharge. Pain is usually present, and more or less dyspnœa appears at a later stage. In epiglottic cases, and in growths springing from the posterior wall of the larynx, there is much pain and difficulty in swallowing, especially when the œsophagus or pharynx becomes involved. Hæmorrhages are more common than in the tubercular and syphilitic varieties of laryngitis, and life may be suddenly terminated from this cause.

Secondary implication of the cervical lymphatic glands does not occur as a rule till after the growth has extended to the pharynx.

The laryngeal mirror will reveal the malignant deposit, often in the form of a warty growth with infiltrating margins on either cord ; sometimes an uneven nodule covered by a dusky patch of mucous membrane, but more frequently a deep, foul, extensive ulceration with infiltrated edges, may be seen extending to the depth of the cartilages. The vocal cord first affected is often paralysed at an early stage.

The course of the disease may be protracted and for months the condition of the patient may exhibit little of the usual cancerous cachexia ; but when deep ulceration sets in, especially when hæmorrhages are frequent and the growth has invaded the pharynx or œsophagus, rapid emaciation sets in, and death soon follows.

Diagnosis.—*Syphilis* and *tubercle* are the diseases to be differentiated from carcinoma of the larynx. The distinguishing characters have already been pointed out in discussing the diagnosis of these affections. In many cases a diagnosis may be long doubtful, but the microscopic examination of a fragment of the suspected tissue, snipped off from the diseased cord, will often solve the problem. When this is not possible, the diagnosis can often only be arrived at by a process of exclusion.

Prognosis.—This is always grave ; but when the patient is seen early, and radical surgical treatment resorted to, a fair prospect exists for a very considerable percentage of cases, and in many of these a return of the disease never occurs.

Treatment.—If beyond the reach of a radical operation when first seen, the palliative measures indicated in laryngeal tuberculosis should be resorted to ; often tracheotomy may postpone the fatal issue for many weeks or months. The best operative procedure in early cases is the removal of the growth, with all infiltrated tissue, by means of thyrotomy or subhyoid pharyngotomy.

LARYNX, PAPILLOMA OF

Innocent growths in the larynx occur most commonly in young subjects ; and, though they may spring from any part of the laryngeal tissue, they are most frequently to be found on the vocal cords. *Papillomata* are more common than all the other benign growths taken collectively. These polypi may be single or multiple, and are supposed to be more liable to occur in singers and public speakers, though they are not to be confounded with the so-called 'singer's nodes,' which consist of hypertrophic or inflammatory thickening of the epithelial tissue of the cords. The laryngeal polypus is generally pedunculated, and hangs or projects

into the interior of the larynx ; it has a warty or granular surface, and consists of hypertrophied papillæ, covered with thickened epithelium, being vascular, and sometimes friable. The fibroid forms, or *fibromata*, consist of connective tissue and numerous blood-vessels, present a red and often cherry-like appearance, are often solitary, may be sessile or pedunculated, and sometimes reach the dimensions of a small plum.

Cystomata, or cystic polypi, occur most frequently on the upper surface of the epiglottis. They are caused by cystic changes in the forms already mentioned, or by obliteration of the ducts of mucous glands, and may reach considerable dimensions.

Other benign laryngeal growths, as angeiomata, lipomata, and myxomata, occur, but they are very rare, and of little clinical interest.

Symptoms.—The most common, and sometimes the only, symptom is the alteration in the voice produced by the growth interfering with the function of the vocal cords. This, from slight huskiness, may amount to complete aphonia. When a large tumour springs from the interior of the larynx, which is rare, suffocation or dyspnœa may at any time supervene ; and difficulty of breathing may also occur from solitary or multiple growths confined to the cords or orifice of the larynx, which are the usual sites. Difficulty in swallowing may arise in epiglottic cystomata ; pain is usually absent, and cough is seldom distressing unless in the papillomata of children.

Diagnosis.—As a rule, this is made clear by the laryngeal mirror ; and the situation of papillomata, which are very generally confined to the commissure or anterior portion of the cord, is a point of much importance in the exclusion of *malignant growths*, which are usually situated about the middle or posterior part of the cord. The history of the case, the age of the patient, and the result of the microscopic examination of a fragment of the tissue removed for the purpose are usually conclusive.

Prognosis.—This is generally favourable, as benign growths often, after a time, cease to increase. The tendency of papillomata to recur, especially in young subjects, must not be forgotten. The ultimate prognosis will depend upon surgical interference, the majority of innocent laryngeal tumours being removable.

Treatment.—When the tumour is causing interference with speaking, breathing, or swallowing, it can and should be removed, except in very rare cases, by an intra-laryngeal operation. The exceptions are large growths below the cords, which may require an external operation.

LARYNX, PARALYSIS OF

The mucous membrane and muscles of the larynx receive their nerve-supply from the vagus, joined by branches of the sympathetic. The superior laryngeal nerves, in addition to supplying sensation to the larynx, innervate the crico-thyroid muscles and, in part, the arytenoid muscle—a thick band of fibres stretching between the arytenoid cartilages posteriorly. The inferior or recurrent laryngeal nerve supplies part of the arytenoid and all the intrinsic muscles of the larynx, except the crico-thyroid, all the motor nerves for the larynx, according to Gowers, being derived from the spinal accessory.

Anæsthesia, or paralysis of sensation, in the larynx, is caused by anything obliterating the function of the superior laryngeal nerve. The anæsthesia may be unilateral or bilateral, complete or otherwise, the mucous surface being insensible when touched by a probe guided by the laryngeal mirror. The lesion may be centric, as in ataxia, hysteria, bulbar paralysis, or other cranial condition ; or it may be peripheral, as not uncommonly met with after diphtheria. The crico-thyroid muscles are paralysed at the same time, so that the cords are not rendered tense on phonation.

Symptoms.—Owing either to motor paralysis or to the anæsthesia, or to both, food enters the larynx, and may find its way into the trachea or bronchi, producing suffocation or pneumonia. Sometimes symptoms of irritation or hyperæsthesia precede the anæsthesia, as may be witnessed in the laryngeal crises of ataxia.

Diagnosis.—The loss of sensation is obvious upon touching the membrane with a probe. The important point is the identification of the centric or peripheral cause, as tabes, general paralysis of the insane, bulbar paralysis, syringomyelia, diphtheria, &c.

The prognosis in the centric cases, except in hysteria (which is a rare cause), is always grave ; in diphtheritic cases recovery is the general rule unless food becomes impacted ; and in syphilitic disease the outlook is favourable.

Treatment.—Careful feeding by means of a soft rubber tube, electrical stimulation, and hypodermic injections of strychnia are useful, along with the remedies indicated for the diseased condition which has induced the paralysis of sensation.

Paralysis of the muscles supplied by the recurrent laryngeal is of prime importance, since, owing to the course of this nerve winding round the aortic arch on the left and its relations with the subclavian artery on the right, the loss of power is often the result of the pressure of aneurisms or tumours

in the upper part of the chest or lower portion of the neck.

The centric origin of laryngeal paralysis must never be lost sight of; the lesion may occur in the nuclei of the vagus, being part of a bulbar paralysis, tabes, syphilitic or other growth. The spinal accessory, before joining the vagus, or the vagus-trunk itself, may be the site of injury or pressure anywhere in its course above the origin of the recurrent laryngeal branch.

The paralysis may be the result of the diphtheritic or other parasitic toxin, or of metallic poisons, as lead, arsenic, &c., or it may be a part of the general peripheral neuritis of alcohol.

ADDUCTOR-PARALYSIS

This is practically always bilateral and the result of functional disturbance, being a common symptom in hysteria, as already described. All the muscles which adduct the cords are involved, owing to which they do not approximate and the voice sinks to a whisper, being lost more or less completely. Usually there is no change in the cough, though cough is not a symptom of the affection, except in those rare cases where the paralysis comes on after prolonged voice-strain, being the result of an acute laryngitis. The laryngoscope shows the failure of the cords to approach each other on attempted phonation, the glottis remaining wide open. The onset of the aphonia in hysterical cases may be very sudden, even occurring in the middle of a sentence, and its disappearance may be equally abrupt. The diagnosis, prognosis, and treatment of this affection are discussed under 'Hysteria.' Sometimes slight adductor paralysis is part of an attack of laryngeal catarrh, in which case the voice, though changed, is not aphonic; it can be produced by an effort.

ABDUCTOR-PARALYSIS

When both cords are affected by the paralysis of their posterior crico-arytenoid muscles, the glottic aperture is closed, or may remain a mere chink, on inspiration, so that laboured, noisy, stridulous breathing occurs, though expiration and voice-characters are normal.

The paralysis, if complete, may suddenly prove fatal at any time. It may be the result of centric lesions, as in tabes, disseminated sclerosis, bulbar paralysis, and cerebral syphilis; it is also occasionally met with in hysteria. Pressure upon both recurrent branches in the neck by large aneurisms and goîtres, or the thoracic pressure of large mediastinal growths, and the influence of the toxins of diphtheria,

influenza, and poisoning by alcohol, lead, and arsenic, may be the cause.

Sometimes the paralysis is incomplete, and may produce no symptoms ; but in progressive centric lesions and in severe toxic cases the adductors finally suffer, and total aphonia results, the cords remaining in the cadaveric position, without any stridor, cough, or dyspnœa, except on exertion. The same condition may supervene from long-standing pressure upon both recurrent laryngeal branches in the neck or thorax, when the paralysis becomes complete.

Unilateral abductor paralysis is generally the result of pressure upon one recurrent laryngeal nerve, caused by an aneurism of the aortic arch, the left branch being much more frequently involved than the right, though this latter nerve is sometimes affected by the pressure of pleuritic adhesions, innominate aneurisms, and occasionally by large aneurisms of the arch. The laryngoscopic examination will reveal the paralysis of the affected cord, which remains motionless in the middle line during inspiration, whilst the opposite one moves normally.

There may be no symptom of any kind, the voice, cough, and breathing presenting no change of character ; but when the paralysis is absolute, some hoarseness or voice-alteration may be detected, and this may become aphonic if the adductors also suffer. When the tumour which presses upon the recurrent nerve also exerts pressure upon the trachea, the respiration will be seriously embarrassed, as described under 'Aneurism' ; but dyspnœa, clanging cough, harsh or falsetto voice, and stridor may be marked on exertion when only one cord is paralysed and no pressure exists on the air-passages below, though the voice, respiration, and cough-sounds be normal in quiet breathing during rest.

Three facts must be borne in mind in the investigation of laryngeal abductor paralysis—viz. that the recurrent laryngeal may be involved by the pressure of growths without the adductor muscles being affected for a long time ; secondly, that bilateral abductor paralysis may arise reflexly through pressure upon one vagus-trunk ; thirdly, that when unilateral abductor paralysis becomes established, the adductor muscles, losing their antagonism, become the seat of paralytic contracture, and the vocal cord is maintained in a position of fixed adduction.

Diagnosis.—The laryngoscope will usually demonstrate the nature of the case as regards the fact of paralysis, but the *cause* should be carefully sought for in the cranium, neck, thorax, or elsewhere. Thus, if the weakness of the muscles be due to *diphtheritic neuritis*, there will always be found evidence of the affection in other nerves ; if the result of *tabes*, the state of the pupils and discs, and the absence of the

knee-jerks, with the presence of ataxia, are characteristic. *Disseminated sclerosis* will betray its presence by the peculiarity of speech, nystagmus, tremor, and other signs. *Bulbar paralysis* will be identified by the loss of power in the tongue, palate, and lips, dysphagia, and dribbling of saliva.

In all the above cases the paralysis sometimes may be bilateral.

Ankylosis of the arytenoids may simulate paralysis, but there will be evidence of old ulcerative lesions in some cicatricial changes about their bases.

Thoracic aneurism should be suspected in every case, especially of unilateral abductor paralysis, in which tabes, disseminated sclerosis, and bulbar paralysis can be excluded.

Prognosis.—This resolves itself practically into the prognosis of the primary cause of the nerve-pressure, and since usually the pressure is not removable, the outlook is generally grave. In the case of enlarged glands and goîtres their removal may be feasible, so that the prognosis in these, and in syphilitic, diphtheritic, and hysterical cases, is favourable. Death from asphyxia may result at any moment in complete bilateral abductor paralysis, unless an early tracheotomy removes the peril, or unless the supervention of adductor paralysis has rendered the respiration free.

In those cases where incomplete paralysis is the result of catarrhal laryngitis or voice-strain, the outlook is always most favourable.

Treatment.—The necessity for opening the trachea promptly is obvious in all cases of advanced bilateral abductor paralysis in which the adductors are not involved, and in which dyspnœa is present. The possibility of there being an obliteration of the air-tube lower down, from the pressure of the same tumour which has paralysed the nerve, must be always remembered before operation is undertaken.

Iodides, combined with mercurials, should be pushed in syphilitic cases, and strychnine hypodermically in diphtheritic paralysis ; rest of the voice in catarrhal or inflammatory cases, and electricity in the hysterical form of the disease, should be persisted in.

LATERAL SCLEROSIS. (See under PARALYSIS.)

LEAD-POISONING, or PLUMBISM

In the present article all reference to acute poisoning by lead is omitted, as this is a subject belonging to the realms of pharmacology and toxicology.

Etiology.—Owing to the numerous uses to which lead is put in the arts and manufactures, and to the almost universal

practice of conveying the domestic water-supply through lead-pipes, the possibilities of its entrance into the human system are innumerable.

In factories where white lead is made, and in potteries where the metal is used as a glaze for earthenware, industrial plumbism is common. Painters, plumbers, glaziers, type-makers, and compositors suffer very frequently. Numerous instances of lead-poisoning have been during late years noticed as following the reprehensible practice of taking diachylon (lead-plaster) with the object of causing abortion.

Accidental chronic lead-poisoning commonly occurs through the water-supply being contaminated in its passage through leaden pipes. Wine or cider which had been stored in leaden vessels has in past times produced severe examples of plumbism; and occasionally the condition has been the result of lead used medicinally, either in the form of ointment and lotion or administered internally for long periods. The worst case of lead-poisoning seen by the writer was in a mechanic hopelessly crippled for life. His occupation had been to finish small metal castings with the file, and, in order to prevent these slipping from his grasp, he used a small slab of sheet-lead, lying flat on his bench. Upon the surface of this platform he steadied one extremity of the object during the filing and polishing, the other extremity being held in his left hand. Small particles of lead, detached from the block, adhered to the fingers and found their way into the alimentary canal during feeding. In plumbers and painters, contamination of the food by unwashed hands is a common cause of lead-poisoning, the cleanly workmen generally escaping.

It seems also certain that lead may enter the system through the skin and respiratory tract. Many cases have been recorded where contaminated snuff has produced plumbism.

Individual susceptibility is a marked feature, and women suffer sooner and more severely than men. Intemperate workmen succumb oftener than teetotalers.

Symptoms.—Generally, *anæmia* precedes the more characteristic symptoms and signs of plumbism. The number of red blood-cells is markedly diminished, and these have been found to undergo a granular degenerative change, which is said to be characteristic. The amount of hæmoglobin does not fall proportionally so low as in chlorosis; nevertheless, the anæmia must be regarded as of the secondary type. A metallic taste is usually felt in the mouth.

Colic is one of the earliest and most prominent symptoms, as is seen from the fact that the affection has been long recognised under the name of 'Painter's Colic.' This symptom shows itself by sudden seizures of acute abdominal pain of a paroxysmal nature, usually referred to the umbilical

region, and nearly always associated with obstinate constipation. The abdominal wall is retracted; pressure sometimes relieves the pain, at other times pressure is intolerable. The pulse is often of high tension during the attack, but not always so, the reverse being sometimes the case; and vomiting occurs frequently.

The cause of the colic is probably due to the direct action of lead upon the plain muscular fibres in the colon or small intestine, as experimental data show that localised rings of contraction are seen in the isolated intestine after the administration of lead. Dixon has shown that this phenomenon may be witnessed after the removal of Auerbach's plexus, so that the action of lead in producing colic is a direct one on the muscle, and not requiring the intervention of the sympathetic ganglia, as formerly supposed.

This direct action of lead on plain muscle is seen also in the almost constant tendency of pregnant women to abort when they become the subjects of plumbism. Increased vascular tension is accounted for in the same way, the metal causing contraction of the plain muscle in the vascular coats, and ultimately thickening of the vessels. The heart-muscle also suffers from the direct action of the poison, and, as shown by Phear, reduplication of the first sound must be regarded as one of the usual clinical signs of the cardio-vascular lesions caused by lead.

A *blue line* upon the gums is a valuable sign of lead-poisoning, appearing in both the acute and chronic cases close to the margin of the gum near the teeth. By the aid of a lens it may be seen to consist of a number of bluish-black points, which are caused by sulphide of lead formed in the papillæ of the gum, as sulphuretted hydrogen is evolved from decomposing particles of food or tartar. It is absent where there are no teeth, and is best marked when the gums are spongy. Patches of similar colour on the tongue and inside of the cheek are sometimes seen opposite to a necrosing hollow tooth. A bluish-black line has been observed, according to Oliver, after bismuth and silver salts have been taken for long periods.

Paralysis.—The selective affinity between muscular and nervous matter and lead produces some of the most interesting signs of plumbism. Muscle poisoned by lead soon ceases to respond to electrical stimulation with the induced current, and its nerves show inflammatory changes, such as are seen in peripheral neuritis. These changes end in proliferation of the fibrous tissue in the nerve-sheaths and degeneration of the fibres, the large cells in the anterior cornua being sometimes affected secondarily.

In lead-workers, the affection of the muscles and nerves is most commonly seen in the bilateral '*drop-wrist*' and

'*drop-foot*' which is produced mainly by paralysis of the extensors; in the upper extremity whilst the extensor-muscles of the fingers and wrist become paralysed and wasted, and the flexors somewhat weakened the supinator longus always escapes, unless the shoulder-muscles become involved. In the leg, the tibialis anticus escapes in a similar manner. Lead-palsy may involve the trunk-muscles, and not rarely are the eye-muscles affected; sometimes the larynx suffers, and occasionally a general paralysis ensues.

The advent of the palsy is usually preceded by a sense of fatigue, pain or tenderness in the muscles, with fibrillary tremors; but the paralysis may appear with marked abruptness in some cases. The muscles rapidly atrophy, and show the reaction of degeneration. Oliver points out that in plumbism with paralytic symptoms there is nearly always well-marked fibrillary tremor of the muscles of the lower half of the face when the patient is asked to show his teeth.

Brain-symptoms are not uncommon, the most formidable being convulsions of an epileptiform character, which may prove fatal, or eventuate in confirmed epilepsy, often associated with partial or complete blindness. Delirium and insanity are sometimes the outcome of this so-called lead encephalopathy, the latter condition remaining permanent. Occasionally (without convulsions) a condition is observed which resembles general paralysis of the insane, from which, however, the patient may in rare cases completely recover; and Oliver has pointed out a type of plumbism which closely simulates ataxia. Optic neuritis and hæmorrhages are common in lead-encephalopathy, and cerebral hæmorrhage is liable to occur when the renal and vascular changes have become established.

There are recorded cases with advanced symptoms of brain-degeneration without any of the metal having been found in the cerebral tissue, though lead is usually found in most of the tissues and organs of the body.

Under brain-symptoms must be mentioned those cases of hemiplegia and hemianæsthesia which occur sometimes without mental changes being observable.

Renal signs are often observed; thus there is a diminution in the output of uric acid, which is supposed to explain the intimate association in some cases between gout and plumbism.

Albuminuria—probably the result of a desquamative nephritis in the early stages, owing to the elimination of lead in the urine, and at a later stage the result of contracted kidney—is common in all long-standing cases. Hence the clinical picture may be that of arterio-sclerosis, with its thickened arteries, slight albuminuria, cardiac enlargement, and renal contracture, with gouty deposits in the joints and eventually cerebral hæmorrhage or uræmia.

Pathology and Pathological Anatomy.—These have been incidentally mentioned in connection with the symptoms. As already stated, the metal finds its way generally into the blood through the alimentary tract, though occasionally the lungs and skin may be the route. It is eliminated by the mucous membrane of the bowel, and, to a slight extent, also by the kidneys. It is found in small quantities in the affected tissues and in all the organs of the body, though occasionally absent from the cerebral substance. The most obvious changes are witnessed in the degeneration and wasting of the striped muscles and the interstitial affection of the peripheral nerves described under 'Neuritis.'

Diagnosis.—The history cannot be depended upon in all cases, as the cause of the poisoning sometimes is beyond demonstration. A blue line on the margin of the gum, with the discovery of lead in the urine, in recent cases, is pathognomonic.

Progressive muscular atrophy is closely simulated in the Aran-Duchenne type of plumbism, in which the small muscles of the hand and thumb are first affected by wasting and paralysis, the differentiating points being the completeness of the reaction of degeneration (in lead-poisoning) and the fact that the wasting of the muscles does not occur before paralysis has become marked. In progressive muscular atrophy the wasting and paralysis begin together and proceed uniformly. Gowers describes the wasting and paralysis as sometimes commencing together and proceeding *pari passu*, as in progressive muscular atrophy. In such cases the diagnosis must depend upon the other signs of plumbism, as colic, Burton's blue line, or the detection of lead in the urine.

Prognosis.—As a rule this is favourable when the patient comes under observation early. In the Aran-Duchenne type, where the wasting sets in along with the paralysis, the outlook as regards the recovery of power in the affected muscles is bad.

Convulsions following on previous mental symptoms are often fatal, and in those who recover from the consecutive coma, as already stated, insanity and incurable epilepsy may supervene. In all cases the prognosis will largely depend upon the susceptible patient abandoning his occupation when this brings him into contact with the metal.

Treatment.—Prophylaxis in the form of the most rigid cleanliness is usually successful.

Sulphuric-acid lemonade is recommended as a prophylactic beverage for those employed in lead-works, but the prevention by scrupulous ablution is obviously more rational.

Iodide of potassium, in doses of about 10 grs. three or four times a day, affords the best means of removing the insoluble

lead-salts from the affected tissues and organs ; but the drug is of little use unless employed with full knowledge of the pharmacology of the poison. When the iodide dissolves the insoluble lead-salts in the tissues by forming some compound salt of the metal which is more soluble, this enters the blood-stream, and a small amount is picked out by the kidneys, but the greater portion is eliminated by the mucous membrane of the intestines, from which it is steadily absorbed again after being changed by the sulphuretted hydrogen in the canal. It is therefore necessary that the eliminated lead should be as rapidly as possible swept out of the bowel before time permits of its reabsorption. Hence frequent purging by sulphate of magnesium must be employed, the insoluble lead-sulphate formed being evacuated in the watery motions. Sulphur-baths and various sulphur-compounds are recommended, but their utility is doubtful.

The colic is relieved by opium and purgatives or large doses of olive-oil ; the paralysis is best dealt with by electricity, massage, and the parenchymatous injection of strychnine.

LEONTIASIS OSSEA

This is a rare condition, also known as Megalocephaly, which is characterised by a general diffuse hyperostosis of the facial and cranial bones, or by the formation of great bossy masses of bone-growth or osteophytes, distributed bilaterally upon the maxillæ, nasals, or frontals.

The disease appears before puberty, progresses very slowly, and usually causes death after middle-age by the pressure or encroachment of the hyperostosis upon the cavity of the skull, orbit, or mouth, or by the destruction of the functions of important nerves.

The deformity when of long-standing can hardly be mistaken, as museum specimens show that the disease is confined to the bones of the face and cranium ; but the possibility of its being confused with acromegaly and osteitis deformans should be remembered in the early stages of the diffuse form. In the leontiasis, or lion-like face, of nodular leprosy the deformity is produced by the diffuse infiltration of the cutaneous tissue.

Treatment is of no avail in arresting the disease, but occasionally relief to neuralgic or other pressure-symptoms may be obtainable by the removal of osteophytic growths which are pressing upon nerve-trunks.

LEPROSY

This is a chronic inoculable disease, caused by a specific bacillus, which leads to the production of neoplasms in the cutaneous tissue, mucous membranes, and nerves, eventuating in various destructive lesions and characteristic deformities. The disease is only met with in the British Islands in rarely imported cases, though in early times it was endemic. It is still widely distributed over the surface of the globe, being constantly present in some parts of Norway, Iceland, Russia, the Balkans, Turkey, India, Arabia, China, Palestine, Egypt, Japan, the Pacific Islands, Central South America, New Zealand, and other regions.

Etiology.—Climate, race, poverty, heredity,[1] soil, and dietary appear to play no part, or a very insignificant one, in the causation of the disease, though Hutchinson still maintains that a diet of salted and decomposing fish is a powerful etiological factor. The disease occurs at all ages, but is extremely rare in very young children.

Contagion.—Much interest and discussion has surrounded the question of how the specific bacillus finds its entrance into the body ; and though a few authorities still maintain that the disease is non-contagious, nevertheless it cannot be doubted that the affection spreads from one individual to another. Nerve-leprosy is certainly less contagious than the nodular variety. The marked diminution in the number of lepers in a community after the introduction of compulsory segregation is almost conclusive evidence of the contagiousness of the malady. The long period of incubation, which may extend to many years, is the obstacle to a convincing demonstration in experiments made by inoculation ; and though the risk of conveying the disease by vaccination is small, Sir William Gairdner's cases are almost conclusive. The case reported by Benson of a death in Dublin from leprosy in a man who had never been outside the British Isles, but who had lived for eighteen months with his brother, who had contracted the disease in India, is absolute proof of the communicability of the disease ; though with improved hygiene the risk of infection is small, unless amongst those in close and intimate relations with lepers. It is supposed that bandages and clothes may convey the disease ; the bacilli are believed to enter the body usually through the nasal mucous membrane. Bassewitz reports a case of leprosy which he believed was communicated from a leprous patient to his attendant by the itch-insect.

Whatever difference of opinion may exist about the channel through which the disease enters the human system, there

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can be none about the cause of the malady being due to the presence of Hansen's leprosy-bacillus, which is not only found in the tissues, but in the secretions from the ulcers and in the mucus and expectoration coughed up by lepers with pharyngeal and laryngeal lesions. It has also been recognised in the urine, seminal fluid, and mammary secretion. The bacillus closely resembles that which produces tubercle ; it is cultivated outside the body with great difficulty, and has never yet been successfully inoculated into animals.

Symptoms.—Two well-marked forms of leprosy have been recognised from the earliest times : (1) the nodular or tubercular form, in which the bacillus chiefly invades the skin and mucous membranes of the nose and mouth ; (2) the smooth anæsthetic or nerve-leprosy, where the bacillus selects the nerve-trunks. A third form is also generally recognised, in which the disease-germ attacks both skin and nerves ; this is commonly recognised as mixed leprosy. These types may remain distinctive throughout their course, but eventually the tendency to run into each other develops ; the form which is mixed from the beginning is the least common of the three.

NODULAR LEPROSY

The first symptoms are liable to be overlooked ; they consist of mild constitutional prodromata of malaise, sweating, vertigo, epistaxis, dyspepsia, and fever. This latter may be smart and ushered in with rigors ; it sometimes lasts for weeks before the appearance of the next sign. This is the presence of an erythematous eruption—the so-called leper-rash—which usually appears on the lobes of the ears, nose, over the cheek-bones and chin, and afterwards on the forearms and thighs. It is of a dull red colour raised above the level of the healthy skin, the individual patches being well defined and exhibiting marked infiltration and some hyperæsthesia.

The shining patches, which vary in size from a shilling to a crown-piece, often entirely disappear, leaving but slight discoloration, but only to return again accompanied by fever and malaise. Ultimately they remain, or, more correctly speaking, on their site appear small red papules, which gradually enlarge and form the true leprous nodules or tubercles, which may remain separate or coalesce into a diffuse infiltration as large as a poached egg. Separate nodules appear upon different parts of the body where no previous erythematous rash had existed. The nodules are shining and tense, of a yellowish-brown colour, and the previous hyperæsthesia gives place to anæsthesia from nerve-pressure ; their distribution varies exceedingly, the entire body-surface being here and there invaded with them—the only regions escaping being the hairy scalp and the glans penis. There is much thickening and infiltration over the elbows and knees, and on the face this

gives the lion-like aspect or *facies leontina* to the leper. The mucous membranes become involved; the conjunctiva, nasal, buccal, pharyngeal, and laryngeal surfaces suffer, and the invasion may extend to the trachea and bronchi, or even to the vagina and uterus. Exacerbations of fever, followed by the deposition of new nodules and glandular enlargement, occur every few months, though absorption of some of the old nodules is often noticed after these attacks.

The nodules may entirely disappear, leaving only a stained area to mark their site ; often, however, some atrophic scarring remains, but more commonly they ulcerate, and in some cases as the ulcers heal, deformities of various kinds ensue. Eventually, however, the healing process ceases, and large tracts of cutaneous surface may become the seat of phagedænic ulceration and foul discharge, which terminate the patient's life by exhaustion, or by renal and pulmonary complications or diarrhœa. Secondary kidney-disease is a very common cause of death.

The hair of the eyebrows is shed early, the nails fall off, the testicles atrophy, sexual power fails, and female lepers are usually sterile. Total blindness is not uncommon from the invasion of the tissues of the eyeballs, and the laryngeal obstruction may cause death. When the nerves become ultimately involved, as they nearly always do, there is pain, succeeded by anæsthesia, and the case clinically resembles the *mixed* form of the disease.

Though recovery is possible, it is extremely rare, and the entire course of the disease may not exceed twelve months, but cases of twenty years' duration are not very infrequent.

SMOOTH LEPROSY,

also known as anæsthetic and nerve leprosy and maculo-anæsthetic leprosy, usually runs a longer and milder course. There may be prodromal symptoms as in the nodular type, but these are as a rule less marked, though neuralgic pains or numbness in various regions are complained of before the appearance of the characteristic leprous spots.

The maculæ appear usually on the back, buttocks, knees, and elbows, but every part of the body-surface is liable to be involved, with the exception of the hairy scalp ; sometimes the spots follow the course of certain nerves. They are not elevated, as in tubercular leprosy, are of the extent of two or more inches in diameter, of a dull reddish-brown colour in white races and of a bright yellow in negroes. Their erythematous character is obvious at this stage by the disappearance of the colour on pressure ; later on pigment is deposited in them, but sometimes on their first appearance pigment may be observed. The pigmentation, however, ultimately disappears, the bleaching process beginning in the

centre, which usually becomes anæsthetic; whilst a ring of colour may surround the spot at its margin, in which some hyperæsthesia always exists. The margin at this stage slowly extends, and is distinctly raised above the level of the surrounding skin. These changes in the maculæ are not usually observable till after the expiration of a year or two. Neighbouring spots may coalesce, so that extensive tracts of the skin are involved, vesicles, pustules, or small bullæ commonly appearing on their gyrate margins. The white atrophied skin in the centres of the spots becomes dry, hairless, and wrinkled; desquamation is constantly present, and the lymphatic glands in the vicinity enlarge. Whilst the above changes have been progressing in the spots, and sometimes even before the spots have been noticed, symptoms of neuritis develop. The ulnar and peroneal nerve-trunks usually suffer first, and anæsthesia of the regions supplied by them becomes marked, as evidenced by the loss of sensation to touch, pain, and changes of temperature; though tactile sensibility may sometimes remain after the disappearance of sensibility to heat, cold, and pain. Severe neuralgic or intense burning pain and hyperæsthesia often precede or accompany the numbness, which latter may extend so as to involve the whole of one or more limbs, or it may affect the mucous membrane of the mouth and throat, and the entire face. The nerve-trunks, where superficial, can usually be felt to be enlarged and tender to the touch.

Paralysis of motion ultimately follows the nerve-destruction. This is often seen to commence in the ring and little fingers, whose tendons contract, and slowly the entire hand becomes claw-like with all the fingers flexed on the palm, and the hand-muscles undergo wasting. The feet and toes suffer in a similar way, and the leg-muscles atrophy.

With the anæsthesia, trivial injuries produce extensive trophic changes, and large bullæ spontaneously appear on the anæsthetic regions and may continue to come out in successive crops for years; they are slow in healing, leaving white superficial scars. These solitary bullæ are characteristic of the late stage of the disease, and contrast with the multiple smaller hyperæsthetic bullæ or vesicles of the earlier period. They often invade the palms and soles, but are very rare on the face.

Deeper lesions ultimately develop: perforating ulcer of the foot and joint-disintegration, like that met with in ataxia, are not uncommon. The bones of the fingers and hand may undergo interstitial absorption, or extensive necrosis may cause their shafts to be shed; and these changes lead to a very peculiar mutilation in which the atrophied peg-like remnants of the nails may be seen attached to the tissues over the carpus or tarsus. Gangrene of a limb may occur and lead to

a spontaneous amputation at the ankle, elbow, or even at the knee-joint. The changes in the facial aspect produced by anæsthetic leprosy are in marked contrast to those seen in the nodular type; they are the result of paralysis of the orbicular muscles and of the parts supplied by the fifth nerve, ophthalmia, ulceration of the cornea, and perforation of the eyeball being not unusual.

The progress of the disease varies considerably. In some cases, after the anæsthesia has spread so as to involve a very large extent of the entire body-surface (which remains white and withered-looking), the stage described as that of *permanency* is reached, in which there may be little noticed save remnants of the eruption, some anæsthesia, and contraction of the fingers. In this condition the patient may sometimes remain for many years, in fair bodily health and with his virile powers undiminished. In a not inconsiderable percentage of cases recovery may be said to have taken place, though some remains of the paralysis may be observed.

The average duration of life after the onset of the symptoms does not fall far short of twenty years, and twice this period is reached in many instances. Death is either the result of gangrene or of the exhaustion caused by extensive ulceration or diarrhœa, the kidney seldom suffering, as is the rule in nodular leprosy, unless the suppuration has been very profuse for a long period.

MIXED LEPROSY

As just described, the variety of leprosy known as the nodular, which commences in the skin, nearly always involves the nerves in its late stages. Nerve-leprosy, upon the other hand, usually maintains its type throughout, though in a small percentage of cases nodules may be observed to be deposited in the skin towards the end. If the term 'mixed leprosy' be retained it should be limited to the clinical type of the disease, in which both the skin and nerve-trunks are involved from an early stage. Such a strictly defined clinical type is rare, especially when it is considered that sometimes, as the disease progresses, the nodulation disappears almost entirely, only the nerve-symptoms remaining.

Crocker regards destruction of the nasal cartilages and ulceration of the soft palate as characteristic features of this type of leprosy. The symptoms and signs of the mixed form are those described already as belonging to the nodular and anæsthetic types, and need not be repeated. Hillis maintains that in a large percentage of the mixed form heredity can be proven, but most modern authorities doubt that heredity is a factor in the etiology of leprosy. Owing to the long latent period, which, as already stated, may extend to many years (certainly it may exceed a quarter of a century), it is

impossible to demonstrate the hereditary transmission of the disease, but probably an hereditary weakness to the leprosy-bacillus may be handed down to the offspring, as in the case of tuberculosis.

Pathological Anatomy.—Hansen's bacillus resembles the tubercle micro-organism ; it is slightly shorter and is somewhat club-shaped, stains more easily, and it shows a marked tendency to develop in groups or colonies. The bacilli are found in great numbers in the nodules, either inside or external to the cells of the tissue, and their presence has been demonstrated in all the viscera, especially in the enlarged spleen and liver and in the endothelium of the blood-vessels, but rarely in the blood itself. The characteristic nodule is a granuloma or new-growth, caused by the irritation of the bacillus, consisting of cells resembling lymphocytes, many of which contain numerous bacilli, though the cells of the epidermis are free from them. Giant-cells occur in the leprosy-nodules, but they are much less numerous than in tubercular neoplasms. In the thickened nerves the bacilli are plentiful in the cells which infiltrate the sheaths, and they are found amongst the parenchymatous elements also. The bacilli are not found in the joints or wasted muscles, these lesions being the result of trophic changes or neuritis.

Diagnosis.—Those acquainted with leprosy agree in stating that the disease cannot be mistaken for anything else when once the symptoms have become established. The writer, from his own knowledge of the signs, cannot speak on the subject of diagnosis, his entire experience being limited to a brief observation on a few cases at Bergen.

Syphilis resembles leprosy more closely than any other disease does. The syphilitic eruption is universally distributed over the body ; the patches are small and but slightly elevated, and they contrast with the large dusky-brown raised patches of erythema, which constitute the leprous spots, with their central anæsthesia and circumferential hyperæsthesia limited at first to some particular locality of the body. In the early stages of leprosy the shedding of the hair of the eyebrows is a valuable diagnostic sign. At a later stage, especially in mixed leprosy, when there is ulceration of the nasal cartilages and palate, syphilis is easily excluded by the presence of anæsthesia, and by the *facies leontina* and other signs ; both diseases, however, are sometimes met with in the same subject.

Nerve-leprosy is likely to be mistaken for *syringomyelia* only because of its anæsthesia. The persistence of tactile sensibility after the disappearance of sensibility to pain, heat, and cold, which sometimes exists also in the leper, may make the diagnosis most difficult. The presence of enlarged lymphatic glands and thickened nerve-trunks, with remnants of

the maculæ, and above all the discovery of the bacillus in a fragment of excised nerve-tissue, will settle the diagnosis.

Lupus is differentiated by its distribution and apple-jelly nodules, its history and progress, and by the absence of the characteristic anæsthesia of leprosy; it is well to remember that both diseases show marked reaction after injection of Koch's original tuberculin.

Prognosis.—As already mentioned in the description of the progress of the special varieties, the disease, though generally incurable and fatal, is sometimes susceptible of complete arrest, the leper escaping with more or less deformity. In a considerable percentage of these apparent cures the bacillus remains latent, and after a long period of many years' quiescence the disease may break out afresh and terminate fatally, or tuberculosis may supervene. The prognosis in nerve-leprosy is more favourable than in the nodular, and in aged than in young subjects, and in temperate than in tropical climates.

Treatment.—This is most unsatisfactory. No true specific remedy has yet been discovered; nevertheless much may be done to alleviate the miserable existence of the leper and to prolong his life. Gynocardic acid, chaulmoogra oil, and Gurjun balsam, used internally and externally, have some modifying influence upon the progress of the malady. Anti-venene and serum-therapy have already given results which may be said to be at least hopeful, and some improvement often follows the use of the X-rays.

Isolation, or segregation, and the employment of every means which improves the general standard of life and increases the resistance of the body-cells to the leprosy-bacillus, must be employed. Removal to a district in which the malady is not endemic is most desirable. Surgical measures are indicated for the removal of the many deformities and complications.

LEPTUS AUTUMNALIS

This is the name given to the harvest-bug or mower's mite—a small red parasite with six legs which abounds in pastures and gardens, especially in limestone localities, near the sea-coast. It may attack any portion of the cutaneous surface, but is found most frequently upon the legs and ankles. It buries its head in the skin by means of its sharp proboscis, causing a smarting urticarial and intensely itchy papule; the itch is further aggravated by the short bristles or hooklets attached to the legs of the insect. In sensitive skins the irritation may spread into large wheals and a severe dermatitis may follow. The parasite is readily destroyed by a strong carbolic or weak sublimate solution, or by a few applications of the pentasulphide of lime solution which is used for common itch.

LETHARGY. (See under HYSTERIA, p. 692.)

LEUCHÆMIA, LEUKÆMIA, or LEUCOCYTHÆMIA

This is a disease of the blood-forming organs, or possibly of the blood itself, characterised by great permanent increase in the number of the white blood-corpuscles, which exhibit changes in their relative numerical proportions, with a diminution in the number of the red cells, and associated with changes in the spleen, bone-marrow, and lymphatic glands.

The disease is not to be confounded with *leucocytosis*—a condition characterised by an increase in the finely granular polymorphonuclear leucocytes; the essential feature in leuchæmia being the increase in the mononuclear white corpuscles, or the presence of myelocytes.

Formerly three distinct types of the disease were recognised, in accordance with that portion of the blood-forming apparatus most obviously at fault—viz. (1) splenic; (2) lymphatic, and (3) medullary or myelogenic leuchæmia. But since the spleen is nearly always found enlarged and altered, and since the bone-marrow is very rarely seen to be the sole tissue at fault, it is considered more satisfactory to recognise only two clinical forms of leuchæmia—viz. the *splenomedullary* and the *lymphatic*.

A classification of leuchæmia founded solely upon the qualitative and quantitative changes observable in the leucocytes has also found favour with clinicians—*i.e.* (1) *myelæmia* or myelocythæmia, in which type the myelocytes characteristic of the normal bone-marrow abound in the blood, and (2) *lymphæmia*, where the lymphocytes found in normal lymphatic glands preponderate. These new terms are practically synonymous with splenomedullary and lymphatic leuchæmia, and they are especially acceptable to those numerous pathologists who believe that the splenic enlargement is a secondary and not a primary phenomenon.

Etiology.—The causation of leuchæmia is still unsolved. Some observers regard the lymphatic form especially as the result of a specific parasite; but there is as yet no evidence to support this hypothesis, though Löwitt has figured objects which he believes to be sporozoa. The disease occurs at all ages, but is more common about middle-life, and males suffer more frequently than females. The acute lymphatic form is most commonly met with in children. A few cases are immediately preceded by some infectious disease, as malaria, influenza, or typhoid fever. Pregnancy, hyperlactation, trauma,

and previous hæmorrhages or anæmia have been suspected as etiological factors.

Symptoms.—There are many symptoms common to both types of the disease ; indeed, in the chronic form of both varieties the constitutional symptoms are practically identical, though the state of the blood shows distinctive features, and there are differences as regards the organs which are the seat of enlargement.

The onset, except in the uncommon acute cases, is usually insidious ; some pallor or anæmia is generally present at an early stage, and ultimately it becomes extreme. The general nutrition of the body may not, however, show signs of deterioration for a considerable period, but at a later stage there is marked emaciation.

Usually the first symptom which attracts the patient's attention is the mechanical effect of the enlarged spleen, which causes a dragging sensation or feeling of weight and a real increase in the girth of the abdomen. Weakness and languor are complained of ; there are rapid action and palpitation of the heart and some quickness of the breathing in proportion to the amount of anæmia present, and bleeding from the nose, gums, or other mucous surface is common. At a later stage purpura-like hæmorrhages into the skin, retina, or cerebral tissue may be observed, and sometimes they prove fatal.

Intestinal and gastric disturbances, as vomiting and diarrhœa, are present, with much thirst and some fever, which latter is, however, liable to long periods of intermission ; occasionally sweating is profuse.

The lungs are, as a rule, free from disturbance, but extensive hæmoptysis and sometimes pulmonary œdema, with great dyspnœa, may be present. The pleura is liable to become the seat of bloody exudations, which are generally bilateral. The urine often contains albumin, especially when the splenic tumour is large ; it is always acid and contains excess of uric acid. Œdema of the legs, ascites, and sometimes general anasarca are frequent late signs. Occasionally priapism has been observed as a prominent symptom, and it may occur early in the disease.

The splenic enlargement is the most common and obvious sign of the disease ; it exists in all the cases of the splenomedullary variety, and at a late stage in the great majority of the chronic lymphatic cases, though usually not to as great an extent. The tumour is smooth, firm, and elastic, occupying a variable extent of the abdomen, usually by its weight sinking downwards and forwards into the epigastrium, and reaching the pelvic brim. When moderately enlarged it may be seen to move freely in respiration, but when greatly increased in size it may cross the middle line, distend the entire abdomen and

become immovably fixed. Upon its smooth blunt anterior margin one or more notches can usually be easily palpated. The tumour is often painless, but more or less tenderness can be elicited by the pressure of the fingers, which sometimes appreciate a well-marked friction-fremitus, whilst auscultation during the act of deep respiration may reveal a creaking sign due to perisplenic inflammatory exudation or adhesions ; occasionally a murmur may be heard over the enlarged organ. The dimensions of the tumour fluctuate from time to time, especially in spleens which have not reached a very great size. The growth may be large even when the blood-changes may not be extensive, and, speaking generally, the magnitude of the tumour depends more upon the chronicity of the case than upon the increased number of the leucocytes, as in acute cases these may be enormously increased with a splenic tumour of moderate size.

The lymphatic glands in the splenomedullary type are but slightly enlarged, and usually only in the late stage of the disease, but very occasionally some groups of them may be found considerably increased in size. In the chronic lymphatic type they are always involved throughout the body, being especially affected in the superficial groups, as in the neck, groins, and axillæ. They form large, painless, elastic tumours, which never suppurate nor become adherent to the skin. The deep lymphatic glands of the thorax and abdomen may exert pressure upon neighbouring organs and cause various symptoms. Lymphatic tissue in other parts, as in the tonsils and Peyer's patches, shows similar hypertrophic growth, and Litten has drawn attention to small lymphatic tumours which sometimes appear under the skin of the eyelids.

The bone-marrow, though found changed so constantly after death, does not give rise to any signs indicative of its implication save by the blood-examination ; occasionally the ribs and sternum have been found tender and painful.

Acute lymphatic leuchæmia occurs chiefly in young subjects, and often runs a fatal course in a few weeks ; it is a rare disease, the chronic form being more common, though this latter is much rarer than the chronic splenomedullary disease. In the acute cases of the lymphatic type the glands and spleen may not have time to become markedly enlarged. There are from the onset fever, rapidly progressive anæmia, and exhaustion, with hæmorrhages into the retina and skin from the mucous surfaces, the blood alone affording conclusive evidence of the nature of the affection. The differential count shows extreme leucocytosis, the predominating cell being the large lymphocyte, though Whipham and Leathem draw attention to a rare type in which the small lymphocytes are greatly increased.

Blood-changes in leuchæmia.—These are by far the most important evidence of the disease and constitute its most characteristic feature. A drop of blood caused to flow from the lobe of the ear or the finger-tip by a needle-prick may be generally seen by the naked eye to be markedly altered, so much so that its watery pallid colour might cause it easily to be mistaken for some other liquid. Under the microscope the number of leucocytes is found greatly increased.

One white corpuscle to about 500 red cells may be taken as a high average in health, and the number of red cells in one cubic millimetre of normal blood should be about 5,000,000, so that the white corpuscles would number in this bulk about 10,000. In leuchæmia the white cells may reach over 1,000,000 ; but since the red cells may in advanced cases fall to little over this number, it is obvious that an equal number of white and red corpuscles might be counted in the c.mm. of leuchæmic blood. Such an increase in the white cells, however, is seldom met with, the white corpuscles generally averaging 1 to 5 or 10 of the red ones ; but the number varies very greatly in the same patient at different hours of the same day, and the proportion of the white to the red cells may for brief periods become normal. The essential point in the blood-examination is the differentiation of the various forms of white corpuscles and the recognition of their relative numbers, as only in this way can the types of leuchæmia be identified.

The blood in splenomedullary leuchæmia.—The white corpuscles in this form reach a higher total as a rule than in the lymphatic variety : the actual number per c.mm. usually ranges between a third and a half of a million.

In stained films the lymphocytes (small mononuclear white cells with rounded nuclei) are, *relative* to the other varieties of white corpuscles, much diminished in number, falling to about a tenth of the whole white count, though there may be an *absolute* increase in their number.

Myelocytes are present to the extent of one-third the number of white corpuscles ; these are large mononuclear neutrophile cells whose faintly staining nucleus almost fills the cell. They are not met with in normal blood, are believed to be produced in the bone-marrow, and constitute the predominant feature in the blood-change in this variety of leuchæmia, though they are also met with in small numbers in some forms of anæmia and in severe septic states. Eosinophile-cells are increased absolutely and also usually relatively. Polymorphonuclear neutrophiles are relatively diminished, but absolutely increased ; these cells form the greater number of the white corpuscles in normal blood, and are supposed to be the descendants of the myelocytes, though in the later stages of the disease they may become diminished in number. Transition-forms between these latter cells and the myelocytes may be

noticed, and cells resembling myelocytes, but with eosinophile-granules, are also to be seen. Basophile-cells, with fine or coarse granules, are also present both in absolute and relative increase ; since they exist in such small amount in normal blood, their presence in splenomedullary leuchæmia is of special importance.

The absence of amœboid movement amongst the white cells is somewhat characteristic of leuchæmic blood, and is in marked contrast to the large number of cells showing this phenomenon in ordinary leucocytosis.

The red cells are diminished often to half the normal number, and the hæmoglobin to a degree greater than the fall in the discs will account for—*i.e.* the colour-index falls below 1.

Nucleated red cells are nearly always seen in considerable numbers ; they are mostly normoblasts or small red nucleated corpuscles, but megaloblasts or gigantoblasts are sometimes, but not often, to be met with.

The blood-plaques are increased considerably, according to Litten and Pruss.

All the above changes may be seen in most examples of splenomedullary leuchæmia, but the differential count varies considerably from time to time in the same case and in different cases. The most constant single characteristic of all the features of the blood is the presence of the myelocytes, or marrow-cells.

The blood in lymphatic leuchæmia.—The most characteristic and marked change is seen in the enormous relative preponderance of the small lymphocytes over the other white cells, though the total white count never reaches the high figures met with in the splenomedullary form. A quarter of a million of white cells would be far above the average in a c.mm. of blood, and of these 90 to 95 per cent. would be lymphocytes of small size, though very occasionally the larger forms may be very numerous. Myelocytes and eosinophiles, generally speaking, may be said to be always absent, the polymorphonuclear corpuscles making up the balance between 95 and 100 per cent. Sometimes towards the end in chronic cases all the white cells are of the small lymphocyte type, only a very few of the polymorphonuclear being visible. The red discs are reduced in number, and as anæmia progresses they fall to less than half their normal amount, and then nucleated red corpuscles may appear in considerable numbers, and the presence of these in abundance along with large lymphocytes may characterise the acute form of lymphatic leuchæmia seen in children.

The colour-index is well below 1, and blood-plaques are scanty.

Whilst the above definite results of a differential count of the white corpuscles are usually quite sufficient to distinguish

the splenomedullary from the lymphatic form of the disease, sometimes mixed types of leuchæmia are met with, in which myelocytes from the marrow and lymphocytes from the lymphatic glands struggle for preponderance.

Morbid Anatomy and Pathology.—The anæmia, wasting, dropsy, and other signs observable during life are obvious in the examination of the body. In addition to the splenic, lymphatic, and marrow changes, the liver is found usually enlarged, pale, and somewhat softened; and the kidneys present the same appearance, the change being due to the infiltration of their cells and connective tissue with leucocytes, hæmorrhages and infarcts being also common. There is usually enlargement of the thyroid, thymus, and the solitary glands and Peyer's patches.

Spleen.—The macroscopic characters of this organ correspond to the physical signs of its enlargement. It is firm and elastic, with its capsule thickened and rough from recent perisplenitis. It may weigh as much as fourteen pounds in chronic cases. On section, it is in acute cases of a deep red, and in old cases of a lighter colour and tougher consistence from hypertrophy of the fibrous stroma. The essential change, however, under the microscope, is seen to be the infiltration of the parenchyma with new cell-formation consisting of enormous quantities of lymphocytes packed closely together in dense patches. The corpuscles of Malpighi are indistinct or invisible amidst the general hyperplasia.

Lymphatic glands.—These, as already stated, are found enlarged along with the spleen in the majority of long-standing cases of both varieties of leuchæmia, but they attain their largest dimensions in the chronic lymphatic form. The glands are soft, smooth, isolated, and movable, and of a pale pink colour, and may reach to the size of a bantam's egg; a clump of enlarged glands may, however, grow beyond the size of a goose's egg. Gulland points out that in the lymphatic type all traces of the glandular arrangement and structure have disappeared, and are replaced by a mass of lymphocytes packed tightly together, obliterating all distinction between the cortex and medulla, germ-centre, and lymph-path. In the splenomedullary variety, though these distinctions maintain, the dense packing is not so marked, being often confined to patches of cells in the cortical parts.

Bone-marrow.—The change in this tissue is certainly the most important of all the pathological features of the disease. There is a very rare type of leuchæmia which has been seen by a few competent observers, in which the lesion has been found to be entirely confined to the bone-marrow, the spleen and lymphatic glands having escaped; to this the term 'acute myeloid leuchæmia' has been given.

In every case of the lymphatic and of splenomedullary form, the bone-marrow is found after death to be diseased.

The most obvious change is noticed in the long bones, whose cavities normally should only contain yellow fatty marrow. Here the fat is replaced by red lymphoid tissue, resembling to the naked eye, and under the microscope, the red marrow, save that the tissue is devoid of fat and is packed with the various neutrophile and eosinophile corpuscles and myelocytes, which give the different varieties of leuchæmic blood their differential characters. In the lymphatic form of the disease the marrow in all the bones, according to Gulland, resembles the structure of the diseased lymphatic glands, the proper tissue being replaced by lymphocytes.

With the changes in the marrow of the long bones there is always also extensive hyperplasia of the red marrow of the flat bones, in which all the varieties of red nucleated cells, eosinophile white corpuscles, neutrophile myelocytes, mononuclear, and polynuclear lymphocytes are found.

The pathogeny of leuchæmia is still awaiting demonstration; the old view of its being a suppuration of the blood, which arose from the pus-like coagula found in the heart, is no longer tenable. The presence of nucleated red cells and myelocytes renders it certain that the marrow-changes play a most important part in the production of leuchæmia, and the theory of Neumann, that all the corpuscular forms met with in the blood are derived from the marrow, gains ground. The spleen-changes are regarded by most authorities to be secondary, the organ merely acting as a reservoir for the leucocytes; and some regard the enlarged lymphatic glands in a similar light, though Ehrlich believes the lymphatic form is essentially different from the splenomedullary, being due to a primary hypertrophic affection of the glands.

The close resemblance of the acute lymphatic variety to ordinary severe infective processes suggests that the prime factor in the disease is a micro-organism which ultimately leads to a mixed leucocytosis by causing changes in the marrow-tissue. As already stated, the so-called parasites described by Löwitt are not believed to be sporozoa.

Diagnosis.—The blood-change, especially that demonstrable by a differential count of stained films, is the only certain method of arriving at a correct diagnosis, especially as leuchæmia may exist with a moderate absolute increase of the white cells. The differential count easily eliminates extensive leucocytosis, splenic and pernicious anæmia, cancer, and Hodgkin's disease; in the acute lymphatic form in children it may be the only method of negativing typhoid fever. It cannot be too emphatically stated that the mere absolute increase of the white cells, unless these reach above a quarter of a million in a c.mm., does not constitute leuchæmia, and it

must be remembered also that during the progress of the malady the proportion of white to red cells may at times fall to normal, especially in the presence of intercurrent affections.

The ophthalmoscopic appearance of the pale orange retina, with its wide bright red veins and white hæmorrhagic patches, is an important diagnostic feature.

Prognosis.—The disease must be regarded as always leading to a fatal issue, though patients believed to be leuchæmic have occasionally been reported as having recovered. The duration in the most chronic examples of the disease rarely exceeds three, and more commonly it falls short of two, years. Periods of temporary improvement in the clinical symptoms, during which the blood-count becomes more favourable, are liable to mislead.

The splenomedullary form is usually of longer duration than the lymphatic, but the acute variety of this latter type in children may prove fatal in a few days or weeks. Hæmorrhages, high fever, severe anæmia, diarrhœa, and especially acute pneumonia, may suddenly terminate life at any stage of either variety.

The prognosis as regards duration of life is not deepened by the degree of splenic enlargement, as this organ reaches its greatest dimensions in the most chronic cases.

Treatment.—Absolute rest of body and mind is essential in the severe forms of the disease ; the dietary should be selected which is compatible with the amount of rest practicable, and it must be one which is easily digested. An animal diet affords the best results, as starches, fats, and sugars are not well assimilated. Of drugs little which is satisfactory can be definitely stated ; nearly every form of blood-tonic and antiseptic has been tried, mostly in vain. The only substance which seems to possess any effect upon the blood-condition is arsenic. Cacodylate of sodium is the best form for the administration of the drug hypodermically. Though it is clear that this substance does not act like the soluble arsenious anhydride, nevertheless, after full doses have been administered the improvement in the clinical symptoms and in the blood-count is such as renders it hopeful that, if employed in a very early stage of the disease, it might prove efficacious.

Bone-marrow extract, thyroid gland, phosphorus, oxygen, and Röntgen rays have of late years been extensively tried. The power of the latter agent in reducing the size of the enlarged spleen and glands, and in reducing the number of white cells in the blood is, according to many reports, most striking, but as the rays increase enormously the metabolic activity of the tissues in leuchæmia, the danger of auto-intoxication in the acute lymphatic form is to be guarded against by insisting upon sufficient intervals between the exposures, as pointed out by Williams.

LEUKANÆMIA

This name has been applied by Leube to a group of cases which present blood-characteristics closely resembling those observed in pernicious anæmia and leuchæmia. Both the leucocytes and the erythrocytes show obvious disorganisation or destructive changes in these cases, which prevent the inclusion of the condition in the group of pernicious anæmia, or in that of leuchæmia. The combination of the destructive changes has also led to the synonym *myeloid splenic anæmia* being applied to the disease. Drysdale, after a careful analysis of the recorded cases, concludes that the majority of them belong to the group of atypical myelocytic leuchæmias, and he considers that the term 'leukanæmia' is a misleading and useless one, and should be abandoned.

LEUCOCYTOSIS

This term, as usually employed, simply means an actual increase in the number of the white corpuscles in a given quantity of blood, regardless of the differential count, though it is the finely granular or neutrophile polymorphonuclear leucocytes which are nearly always found increased. These exist in normal blood in the proportion of about 65 to 75 per cent. of the entire white cells present.

As already stated, 10,000 white cells per c.mm. is a rather high average for normal blood, but after a full meal, during pregnancy, and in the first month of infant life a physiological leucocytosis is the rule, which may extend considerably beyond this standard.

In the examination of the blood in diseased conditions, it may be accepted that a count which shows the presence of as many as 15,000 leucocytes may be accepted as an instance of leucocytosis, and it is rare that more than double this number is to be met with, excepting always in leukæmia, which does not belong to the category of ordinary leucocytosis.

An absolute increase in the white cells is a constantly observed phenomenon in most acute inflammatory affections, as pneumonia, erysipelas, scarlatina, cerebro-spinal fever, acute rheumatism, and pleuritis; and in most suppurative states, as pyæmia, empyema, septicæmia, appendicitis, internal abscesses, small-pox, and phthisis. In malignant disease a considerable degree of leucocytosis is the rule, which increases as the cachexia advances, and the condition is observed as a transient phase in all hæmorrhages. The polymorphonuclear cells with neutrophile-granules, whose increase is the essential feature in leucocytosis, are derived from the finely granular myelocytes produced in the bone-marrow.

It is supposed that chemical substances in the blood manufactured during the suppurative process, and toxins derived from bacterial processes, exert a chemiotactic action upon these cells, attracting them into the blood-stream.

A form of leucocytosis in which the increase is in the eosinophiles is met with in asthma, trichiniasis, ankylostomiasis, and other worm-diseases, and in various skin-affections, as urticaria and pemphigus.

Leucopenia—the opposite condition—is comparatively rare. The term is used to signify the state of the blood in which the absolute number of white cells is diminished. It is, perhaps, best seen in kala-azar and typhoid fever, where the count may fall to about half or one-third of the normal. Pernicious anæmia, chlorosis, influenza, splenic anæmia, and purpura are conditions in which more or less leucopenia is usually present.

In diseased states characterised by marked leucocytosis, as in pneumonia, the disappearance of the excess of leucocytes, especially if to the extent of producing leucopenia, is often a grave sign. Muir points to the truth of the converse of this—the presence of leucocytosis, where leucopenia had been present, as in typhoid fever, being an indication of some secondary complication. In this disease it may be well to remember that, as the diminution is in the finely granular cells, there is therefore a relative increase in the small lymphocytes ; this latter condition is sometimes incorrectly spoken of as a lymphocytosis. A true lymphocytosis is, however, sometimes met with in the tubercular adenitis of childhood and in pertussis.

LEUCODERMIA

This condition, also known as Vitiligo, consists in an anomalous distribution of the skin-pigment, exhibiting itself in scattered patches of white skin with irregular convex outer borders, in which much pigment is deposited.

The affection is much more common in tropical countries and in the darker-skinned races, but it is not rare in Great Britain. It occurs before middle-life, in both sexes equally, and is believed to be more frequently met with in neurotic subjects who have undergone severe mental strain or grief.

The milky-white patches of affected skin are more common about the neck, but may appear upon any part of the body ; they are usually more or less symmetrical, and gradually extend by a shifting of their external border, which is always convex, until in some cases a very large proportion of the entire cutaneous surface becomes affected. The healthy skin close to the margin of the patches is hyperpigmented and gives the idea to the observer as if all the pigment had been

removed from the white patch and been deposited in its abrupt scalloped border, which gradually fades into the tinge of the surrounding normal skin.

The bleached, smooth, supple skin is normal in every respect save in its colour, and its hairs are also whitened.

The change may be limited for an indefinite time to a small region of the skin, but its natural tendency is to slowly progress for many years; and when the entire cutaneous surface has been thus invaded, in some cases the disfigurement passes away, the affected skin being left uniformly whitened and all piebald deformity abolished. Even when the area of skin affected is but moderate, the patient is apt to regard the bleached patches as the healthy parts.

The pathology of the affection is clearly explained on the theory of some alteration in the peripheral nerve-supply of the part, whereby the pigment or melanin formation and distribution is interfered with.

Diagnosis.—The characters of the abrupt hyperpigmented border, which always presents a convex margin on the side merging into the unaffected skin, distinguishes the condition from the more common *chloasma uterinum*. *Anæsthetic leprosy* presents white spots of skin which might be mistaken for leucodermia, but the absence of anæsthesia in the latter is at once distinctive.

Prognosis.—As the question is but one of the extent of the deformity, this will depend upon the region affected and the rate of its progress. The more thoroughly progressive the affection is in some cases, where the face, neck, and hands are involved, the better the outlook—a standstill meaning permanent unsightliness.

Treatment.—No agent has any marked effect upon this condition. Attempts to cause pigmentation in the white areas by blisters and other irritants are useless; and scarcely more hopeful is the plan of reducing the pigment in the dark borders by corrosive sublimate and peroxide of hydrogen solutions, or by the permanganate method, though very occasionally a slight alleviation of the deformity may thus be accomplished.

LEUCOPLAKIA

This is a condition of the tongue which is characterised by the presence of white patches, usually raised above the level of the surface, and which are the result of hypertrophy of the papillæ and thickening of the epidermis. The affection is so frequently met with in heavy smokers that it is known as 'Smokers' Patch'; it has also been described as Psoriasis and Ichthyosis of the tongue, Keratosis, Tylosis, Leucoma, and other fanciful titles.

Etiology.—The causation is certainly some direct irritation, as that produced by the superheated current of air as it strikes a spot on the dorsum of the tongue in being drawn into the mouth in smoking light tobacco. On the inside of the cheeks similar patches may be produced by the irritation of ill-fitting artificial teeth or carious stumps, and these may also affect the edges of the tongue or inside of the lips.

Men are more frequently affected than women, and a small percentage of the cases is believed to be the direct result of syphilis.

Signs.—The white circumscribed pearly patches stand out from the surrounding healthy tongue-surface in a way that can hardly be mistaken for any other condition. The patient, after the discovery of the patch, which he usually regards as a wart or corn, not uncommonly complains of some subjective symptoms of various kinds. In several cases under the writer's observation these were obviously caused by the dread of cancer, and the most of them eventually developed into a genuine epithelioma.

The progress of the affection is chronic, unless the cause be removed early, and it appears to be aggravated by attempts at peeling off the warty cap of the growth. Often this is spontaneously shed, leaving a tender deep red surface, upon which the thickened epithelium again forms. The pearly-white appearance of the patches, and the absence of any tendency to ulcerate, distinguish them from *tubercular nodules*; their density, circumscribed oval or circular outline, and their usual site on the dorsum of the tongue, differentiate them from the mucous patches of *secondary syphilis*.

Prognosis.—The danger of epithelioma supervening must not be lost sight of in all chronic or long-standing cases. When seen early, the prognosis is good if the patient entirely leaves off smoking and drinking strong alcoholic liquors.

Treatment.—As above indicated, the first step is the removal of any obvious cause. Decayed teeth should be removed, and artificial teeth-fittings should be perfected, so as to remove all cause of direct irritation. Hot food or liquids, and spices or stimulating sauces, must be forbidden. Strong iodide of potassium solution (1 in 5), painted over the patch two or three times a day, sometimes causes its speedy disappearance, but generally it resists all drug-treatment. Caustics should not be applied, and when the patch remains, or continues to re-form after it has been spontaneously shed, excision by the knife or galvano-cautery should be resorted to.

LICHEN

This term was formerly loosely applied to most skin-eruptions exhibiting papules, which are now by dermatologists transferred to their own groups, as defined by the nature of the pathological process in each case. Thus, *Lichen urticatus* is recognised as a papular variety of urticaria ; *L. circumscriptus*, *L. circinatus*, and *L. serpiginosus* are forms of seborrhœa ; *L. simplex* is but the papular stage of eczema or impetigo, and when severe or extensive is sometimes still described as *L. agrius*. *L. pilaris*, with its spiny plugs projecting from the mouths of the hair-follicles, is now recognised as a keratosis of the upper part of the follicle ; *L. hæmorrhagicus* and *L. lividus* are the result of minute hæmorrhages in the region of the hair-follicles, occurring in purpuric or other blood-conditions.

L. scrofulosorum, though it remains for a long time as a true papular eruption, each papule being capped by a fine scale, sometimes terminates in a pustule, and is often associated with *eczema impetiginosus*. It occurs so constantly in scrofulous subjects that its tubercular nature, though not demonstrated, is strongly suspected. Since its pathology is obviously an inflammation of the hair and sebaceous follicles, it cannot be regarded as a true lichen. *L. tropicus*, or prickly heat, is a sweat-rash allied to *L. strophulus*, or teething rash, and is caused by plugging of the orifices of the sweat-ducts.

LICHEN PLANUS

After the elimination of the above heterogeneous affections there remains the true lichen, known as *L. planus*, or *L. ruber planus*, an eruption consisting of small isolated or clustered round or polygonal red burnished papules, which may remain isolated or fuse into patches of various size.

Etiology.—Little is definitely known as regards the causation of the disease, which affects both sexes about equally between the ages of twenty-five and fifty years, being comparatively rare in infancy and early childhood. It is more common amongst the better classes, and in a small percentage of the cases there is a history of prolonged mental strain or of neurotic inheritance, which has given rise to the belief that the affection is of nervous origin. No parasite has yet been demonstrated in the papules, and the disease is certainly not contagious.

Pathology.—The changes observed are of an inflammatory nature, confined to the superficial layer of the corium and the papillæ, beginning in the vicinity of the blood-vessels of the latter. The sweat-ducts and the hair-follicles, in their upper portions, are also involved. The papillary layer

becomes extensively infiltrated with small cells, derived from the connective-tissue cells. These produce a solid œdema, which flattens and alters the shape of the papillæ very considerably; the epidermis shows hyperkeratosis of its horny layer, which leads to the formation of horny plugs pressing upon the surface of the papillæ below, and the tension of the horny stratum above produces the glossy burnished appearance of the papule. At a later stage, the hypertrophic changes give way to atrophic or degenerative processes, with the deposit of pigment.

The white milky network so characteristic of true lichen is produced by hyperplasia of the granular layer, and the depression or pit often visible in the centre of the papule is probably the result of the shedding of the horny plug from the mouth of the sweat-duct.

Symptoms.—The papules may appear upon any portion of the skin, the more common sites being the front of the wrists, forearms, and thighs, which are generally involved in a more or less symmetrical manner, not rarely following a linear distribution suggestive of the course of the nerve-supply. The face and hairy scalp usually escape; those portions of the trunk subjected to pressure from clothing or tight-lacing frequently exhibit the eruption in a marked manner. The mucous covering of the palate, cheeks, and tongue also often presents flat white spots or patches corresponding to the elevated cutaneous papules. The palms and soles may be affected, though the papules here do not show as on the other regions, owing to the thickness of the horny layer, and the nails escape.

The papules are at first pinkish, afterwards becoming bluish-red; they vary in size from that of a pin's head to a small split pea, and are usually flattened, being only slightly raised above the level of the skin. The outline is circular, polygonal, or oval, and the surface is smooth and burnished, with often a minute depression in the centre; when they form round hair-follicles they are pointed or acuminate. A fine network of white lines shows through the papule when the horny layer is stretched, and more or less pigmentation is visible in long-standing cases. The papules may remain discrete; but they usually cluster, forming patches of a variable size and outline, which become scaly. This scaliness or hyperkeratosis, especially on the legs, may give to the patch the appearance of a warty or verrucose eruption, but on examination isolated smooth or slightly scaly papules may be discovered outside its borders.

The patches may form rapidly, running an acute course, ending in a few weeks in resolution, leaving behind much dark pigmentation; relapses or recurrences are, however, common. The acute attacks generally involve an extensive

area of the cutaneous surface, which shows small bright red isolated papules and patches of various dimensions. The chronic form of the disease is generally localised, and the warty or nutmeg-grater patches on the legs run a very slow course. Occasionally an acute general attack supervenes upon a chronic localised affection.

Itching is an almost constant symptom, though it varies greatly in intensity. Sometimes it may be so severe in the acute cases as to produce total insomnia and reduce the patient's strength to an alarming degree; in other cases only a slight tingling sensation is complained of. Diarrhœa is sometimes severe, and its cause is supposed to be due to the eruption involving the intestinal surface as it does the inside of the mouth and pharynx.

As already stated, when the hair-follicles are involved the papules become acuminate, and in a form of lichen, first described by Hebra as *L. ruber acuminatus*, all the papules were of this pointed character; the disease ran an acute course, and generally ended fatally. Unna has described under the term *L. neuroticus* a variety of lichen which had been observed to occur in an epidemic form in Hamburg. It was associated with intense itching, nervous prostration, and fever. The papules, however, were preceded by patches of erythema, and the issue was usually fatal. Many authorities, including Brooke, consider that this *L. neuroticus* is the lost type described by Hebra; whilst other dermatologists consider that Hebra's description applies fo *Pityriasis rubra pilaris*.

Diagnosis.—The characters of the papules as above described in the great majority of cases are usually sufficient to establish the diagnosis, and the deep pigmentation which surrounds or marks the site of the patches caused by aggregation of papules is also characteristic.

Psoriasis is distinguished by the scaly nature of the diseased patch from its first appearance, its silvery aspect, the absence of itching, and the favourite situation of the eruption on the elbows and knees. The red bleeding base which is visible when the scales of a psoriasis-patch are forcibly removed is also characteristic.

Eczema, in the papular stage, may occasionally resemble lichen, but the tendency of the dull red eczematous papule to change into a vesicle, which bursts and discharges its serous contents by continual weeping, is quite distinctive.

Prognosis.—Except in the very rare *L. ruber acuminatus*, the disease ends always in resolution under appropriate treatment, though the duration of the hypertrophic horny variety met with on the legs may defy treatment for many months.

Treatment.—This is practically identical with that of psoriasis, and may be summed up by stating that arsenic

internally, in full doses, and stimulating tar-preparations externally, give the speediest and most satisfactory results. Menthol may be combined with the tarry applications when the itching is very severe, and weak sublimate baths are often efficacious. The X-rays have been recently successfully applied to the treatment of the disease, and have been followed, even in chronic cases, with surprising amelioration or cure.

LIVER, ABSCESS OF

Suppuration of the hepatic tissue, or suppurative hepatitis, is said to be either primary or secondary. The terms are unfortunate, since so-called primary abscess of the liver is always secondary to disease of neighbouring organs, which by direct extension implicates the hepatic parenchyma, as in ulceration of the gall-bladder or stomach. The secondary abscess, on the other hand, is one which is induced by some morbific state of the blood which supplies the gland.

Etiology.—All abscesses of the liver may be practically regarded as of parasitic origin, the micro-organisms finding their way by the portal vein, or directly reaching the liver through the ulceration of the gall-bladder or biliary passages caused by gall-stones, or through a purulent cholangitis, or, as in many cases, infection is carried in the arterial supply to the organ.

In this latter case the various pus-forming organisms which produce ordinary pyæmia through the infection of wounds are brought to the liver by the hepatic artery. In the same way the bacilli or cocci which produce malignant endocarditis or pulmonary gangrene are showered into the arterial circulation and swept onwards till arrested by the hepatic capillaries, which they block, and ultimately suppuration or necrosis of the hepatic parenchyma supervenes. Thus in pyæmia these multiple metastatic abscesses are so commonly found in the liver that they are termed pyæmic abscesses.

A large percentage of all cases of suppurative hepatitis or abscess originate in infection carried by the *portal vein*, which receives its infective organisms through ulcers or abrasions in the intestinal and gastric surfaces, from the foci of typhlitic, appendicular, uterine, and pelvic inflammations. Inflammatory states of the gall-bladder and biliary passages, with or without gall-stones, infect their own veins, which open into the portal vein, and may thus become the origin of an hepatic abscess. In the same manner, inflammation of the splenic vein or any of the branches of the portal system may produce a similar result.

This condition is usually described as Portal Pyæmia, Pylephlebitis, or Porto-pyæmic Abscess, and the majority

of the causes above mentioned produce a number of small abscesses studded throughout the hepatic parenchyma.

The intimate connection between dysentery and hepatic abscess has been long recognised, but it appears almost certain that the bacillus discovered by Shiga, and maintained by him to be the cause of acute dysentery, is not alone capable of producing hepatic abscess. This condition is much more common in the chronic or amœbic dysentery, the amœba being itself capable of inducing suppuration or of conveying pus-forming bacilli or cocci to the liver. The abscess so produced is, however, single or solitary, and since it occurs most frequently in Europeans resident in the tropics it is commonly known as the *tropical abscess*.

How far the tropical abscess is to be regarded solely as of dysenteric origin is a difficult question, since it is sometimes found independent of any history of dysentery ; and most authorities believe that other causes than the amœba are at work in its production, though the parasite is so frequently found in the pus. Free living and the use of alcohol, combined with the influence of tropical temperature, are probably important factors, since it is rare in dysentery occurring in natives and rare in Europeans suffering from dysentery in temperate climates.

The difficulty is not lessened by the consideration that the solitary nature of the suppuration is decidedly against the theory of its pyæmic origin, and by the fact that true multiple or pyæmic abscesses also occur in a variable proportion of dysenteric cases.

Traumatic causes account for a small percentage of liver-abscesses, the infective germs finding their way directly through the wound in the abdominal walls to the exposed or injured organ. In a similar way the injuries to the stomach or intestines caused by fish-bones and the wounds produced in rectal operations afford an entrance for infection.

Parasites, as hydatids, actinomyces, and ascarides, may be the carriers of micro-organisms, and sometimes abscesses result from infection of the stump of the umbilical vein in infants.

Of considerable theoretical interest are the rare cases where infection enters through the hepatic vein, being conveyed from some of the veins of the lower extremities, and by stasis in the vena cava inferior the blood is forced during a violent respiratory act into the hepatic vein.

PYÆMIC or MULTIPLE HEPATIC ABSCESS

As already stated, this is but a manifestation of the general pyæmic process arising from the infection of wounds by pus-forming microbes, which, finding their way into the systemic circulation, produce hepatic abscesses by blocking

of the hepatic capillaries, and subsequent necrosis. The microbes found are streptococci, bacillus coli, pneumococci, staphylococcus pyogenes aureus, and others.

The condition is of little clinical interest, since very often it is only recognised at the examination after death, the general symptoms of the pyæmia entirely overshadowing the hepatic abscess. The liver is enlarged, softened, greasy, and riddled with small abscesses, whose walls are formed of the friable hepatic parenchyma. The larger cavities may be as large as an egg or small orange, while the smaller ones may not exceed a large pin's-head in size. The lungs are almost invariably affected in a similar manner, and far more frequently than is the liver. Any of the previously mentioned pyogenic organisms may be found in the pus evacuated from one of the abscesses.

The appearances are identical in *portal pyæmia*, in which the infective germs reach the liver from gastric, intestinal, and typhlitic ulceration, and from infective inflammations of the biliary passages, the only difference being that the abscesses in these cases are confined, as a rule, to the liver, and are not found in the lungs, heart, joints, or muscles.

In some dysenteric cases the abscesses are multiple and of the true pyæmic type, though the large solitary, or so-called tropical, abscess is much more common in amœbic dysentery.

The portal vein is often found the seat of pylephlebitis, pus and decomposing septic clots being visible in its branches or roots.

Symptoms.—These are such as are usually met with in pyæmia, as rigors, sweating, and high irregular fever; and in the general pyæmic cases there is the history of a wound, or evidence of ulcerative endocarditis. Some pain, tenderness, and uniform enlargement of the liver may be discovered, but often the abscesses are not suspected till revealed at the autopsy. Marked jaundice is rare, though a muddy icteroid tinge of the skin is commonly present.

The disease runs a rapid course, and is invariably fatal, the issue being seldom protracted beyond a few weeks.

The symptoms in portal pyæmia are, as a rule, less acute, and the constitutional depression and fever often less marked, so that the local signs of multiple hepatic abscess are often more obvious than in general pyæmia. The first point of importance in such cases is the consideration of the primary affections liable to lead to portal pyæmia. Of these, the infective nature of inflammations in the region of the appendix should not be lost sight of. Ulceration of the gall-bladder or biliary ducts, or infective cholangitis will usually be associated with symptoms of hepatic colic or obstructive jaundice; and the supervention of pain in the right shoulder and hypochondrium, with irregular fever, rigors, and serious

constitutional symptoms of great prostration, passing into delirium or coma, should direct attention to the liver. The organ will be found uniformly enlarged and painful, but fluctuation can seldom be demonstrated, though careful percussion may in some instances afford evidence of the lobe which is most seriously involved. Peritonitis or pleural irritation may develop so as seriously to interfere with respiration, and there may be vomiting, smart diarrhœa, and profuse sweating. When such grave deepenings of the symptoms occur in dysentery, the formation of a pyæmic abscess of the liver may be strongly suspected, and their supervention after rectal operations and in ulceration of the stomach, simple or cancerous, tell the same tale.

Should the infective agent be so intense or virulent as to produce an extensive pylephlebitis of the portal vein, the fever, chills, sweating, and typhoid condition may become as severe as in ordinary general pyæmia, and death may rapidly supervene; or, if the patient live long enough, metastatic abscesses in other organs may be found, proving that the portal pyæmia has ended in a general blood-infection.

Diagnosis.—As already stated, multiple pyæmic abscesses of the liver often remain undiagnosed during life. When the local signs of hepatic pain and enlargement direct attention to the state of the organ, the history of the case and an investigation into the preceding symptoms will often enable the physician to discriminate between a generalised and a portal pyæmia.

From *tropical* or *solitary abscess* the multiple form may be differentiated by the comparative mildness of the constitutional symptoms, the localised nature of the pain and hepatic swelling, the presence of fluctuation, and the more chronic course of the disease in this latter affection.

Gall-stones often give rise to fever, rigors, and sweating, the condition being mistaken for pyæmic abscesses; but the absence of grave constitutional symptoms during the lengthened intervals of apyrexia, and the chronic course and intensification of the jaundice after each attack, are distinctive.

Sub-phrenic abscess can only be eliminated after a painstaking investigation of its history.

Malaria is excluded by absence of the regularity of its intermittent pyrexia, by the failure of the affection to yield to quinine, by the absence of the grave pyæmic symptoms in the intervals between the attacks, and by the results of the examination of the blood for the malarial parasite.

Prognosis.—Pyæmic abscess is almost invariably fatal, but the forecast in abscesses arising from infection of the portal tract should be very guarded; some cases commencing with symptoms suggestive of portal pyæmia ultimately exhibit the characters of a solitary or tropical abscess, which

is curable. This is not a very unusual termination of gall-stone and perityphlitic cases.

Treatment.—The only remedies of any value are those which are mentioned under 'Pyæmia,' by which the fever may be modified and the patient's discomfort alleviated. Surgical procedures are contra-indicated in multiple abscesses, unless an abscess points or threatens to burst into the peritoneal cavity.

SOLITARY or TROPICAL ABSCESS

As implied in its title, this form of suppurative hepatitis eventuates in the formation of one large abscess-cavity in the liver; but the term is somewhat misleading, since in about one-fourth of the cases, there are two or more large abscesses, and, moreover, the condition is occasionally met with in temperate climates.

Etiology.—The association of the solitary abscess with amœbic dysentery, and its relations with that disease, have been already discussed; but it must not be forgotten that the true multiple pyæmic abscess the outcome of portal pyæmia is also met with in connection with dysentery.

Climatic conditions independent of dysentery certainly play an important *rôle* in the production of this form of suppurative hepatitis, since it is frequently met with in tropical and sub-tropical countries in Europeans who have never suffered from dysentery. Alcoholism and over-indulgence in animal food are etiological factors, probably acting in conjunction with dysentery and exposure to high temperature in most cases, and men are more frequently affected than women.

Injury, especially when associated with the etiological factors just mentioned, is an undoubted cause of the solitary abscess, and in cold or temperate climates it supplies the explanation of a considerable proportion of the cases of this comparatively rare condition. The effect of blows or severe contusions in diminishing the vital resistance of the hepatic cells to microbes finding their way into the organ from the intestine is as obvious as is the supervention of tubercular lesions of the spine and testicle following contusions of these parts.

Cholangitis of the suppurative type produced by gall-stones, or the variety which occasionally follows typhoid fever, pneumonia, and cholera, may lead sometimes to the formation of one or more large abscesses, though these conditions are more prone to eventuate in the multiple abscesses characteristic of portal pyæmia. The same remarks apply to gastric, intestinal, or rectal ulceration, and still more rarely to typhlitic inflammation.

Suppurating hepatic hydatids are an occasional cause of the solitary liver-abscess, as are also lumbricoids, which on creeping up the bile-ducts, may carry intestinal micro-organisms into the biliary passages.

Pathological Anatomy.—Though the large solitary abscess in its typical form differs widely from that of the multiple pyæmic abscesses, still many intermediate varieties are met with, which have led some pathologists to regard the pathological processes as differing only in degree, owing to the virulence of the infective microbes and the vital resistance offered to them.

The solitary or tropical abscess is commonly found deep in the convex portion of the right lobe ; it is at first bounded by the ragged, reddened, and softened liver-parenchyma, but gradually a pyogenic membrane forms as it becomes encapsuled and shut off from the hepatic structure by granulation-tissue. The contents, in addition to pus, may exhibit bloody or bile-stained characters, and various microbes have been identified, as the amœba of dysentery, staphylococci, and bacillus coli, but in some instances sterile pus may be alone present.

The abscess may be as small as a walnut or as large as a cocoa-nut ; occasionally it may reach the dimensions of a fœtal head and contain one or more pints of pus. One or two additional abscess-cavities may be found in other portions of the organ. The hepatic tissue in the immediate vicinity of the abscess is usually softened and unhealthy, but in chronic cases the liver-parenchyma outside the dense fibrous capsule of the abscess may be quite normal. Occasionally a firm cicatrix or a calcareous deposit has been found to mark the site of an old abscess whose contents have been absorbed.

The liver itself is always considerably enlarged in acute cases, and evidence of peritonitis and adhesions of various degrees of firmness are present according to the site of the hepatitis.

In the abscesses of amœbic dysentery the contents are often of a viscid consistence, leucocytes are notably sparse, and there are usually diffuse inflammatory changes observable throughout the hepatic parenchyma, caused by the irritation of the toxins manufactured by the parasite. The abscess, owing to its common site in the convex part of the right lobe, is prone to extend upwards and involve the right lung or pleura, hence pulmonary abscesses are not uncommonly caused by the direct extension of the suppurative process. Rarely does the abscess burst into the peritoneal cavity or pericardium ; sometimes it discharges its contents into the transverse colon or stomach, or it may reach the cutaneous tissue and discharge its contents externally at a distance from the liver-region, leaving tortuous fistulous passages. In

a few cases it may form renal adhesions and eventually burst into the pelvis of the kidney ; the hepatic vein or inferior vena cava may be perforated.

Symptoms.—Fever is seldom if ever absent, except in chronic cases. It is often the first symptom, and varies considerably in its intensity. It is always of an irregular or intermittent type, which in malarial regions has frequently led to errors in diagnosis ; but when watched for some days its departure from the recognised types of malaria is obvious. Usually there are evening exacerbations, but these are irregular and often are not preceded by rigors, though followed by profuse sweating. The temperature falls usually, or becomes very irregular, after the formation of the abscess, passing into the hectic type observed in phthisical cases. In acute cases the rigors and sweatings may be as severe as those seen in general pyæmia, whilst in chronic abscess there may be no fever at any stage.

Pain may appear before the onset of fever ; usually it accompanies it. It is felt in the hypochondrium or epigastric region, and when invading the right shoulder it is a highly suggestive symptom. When the hepatic capsule is inflamed in superficial abscesses there is intense pain and much tenderness, contrasting with the dull aching feeling complained of in deep suppuration.

The liver enlarges at an early stage, the swelling presenting a soft feeling when the fingers are applied to its lower rounded margin. The increase in size is commonly confined to the right lobe, which enlarges in an upward direction, elevating the diaphragm and often producing obvious bulging of the side. The liver-dulness is increased in the right nipple-line and more so in the axillary line, and may extend as high as the third rib or downwards in large abscesses towards the iliac crest. Sometimes the left liver-lobe alone exhibits increase in its vertical dimensions, but it is well to remember that extensive enlargement of the right lobe may cause displacement and apparent increase in the size of the left.

Fluctuation can be detected early when the abscess is superficial or on the lower surface ; but this sign is not usually obvious, when deep deposits exist in the right lobe, till after the expiration of a considerable period.

Jaundice is very seldom a marked feature, but more or less icteroid tinting of the skin and conjunctiva may be observed in most cases. Digestive disturbances, as anorexia, nausea, and vomiting, are common ; pulmonary symptoms, as frequent painful coughing, or signs of pleuritis, indicate the extension of the suppurative process to the thoracic contents.

The patient generally lies upon his back, rarely upon either side, owing to the tension of the liver-capsule being increased by the traction of its weight in right or left decubitus.

Sudden amelioration of these signs and symptoms may set in by the evacuation of the pus through a bronchial tube or other route. The expectorated pus is usually of a dark reddish or chocolate colour, and in dysentery active amœbæ have been demonstrated in it.

The duration is variable. As already stated, the progress of the solitary abscess is much slower than that of the multiple pyæmic form; but instances occur in which a fatal issue has supervened within a couple of weeks; more frequently the abscess, when not interfered with, runs a slow course of two, three, or more months. When spontaneous rupture in a favourable direction follows, recovery may be protracted for some years, during which more or less suppuration may be continuous.

Diagnosis.—The irregular fever, chills, and sweating, with hepatic pain and unsymmetrical enlargement of the organ and the detection of fluctuation in the bulging part, are more or less characteristic; but the diagnosis can only be rendered certain by the introduction of a fine needle attached to a hypodermic syringe or aspirator. In considering the question of diagnosis, the main point is to search for the causes which are known to be productive of suppuration within the portal region; in this way the multiple may often be differentiated from the solitary abscess. *Malaria* is differentiated by the regular type of its pyrexia, by the discovery of the parasite in the blood, and by its speedy yielding to quinine.

Empyema of the gall-bladder may be mistaken for an abscess projecting from the lower surface of the liver; but the pear-shaped outline, the ease with which it can be moved from side to side, and the frequency with which a history of hepatic colic may be elicited, are distinctive.

Empyema, abscess of lung, pleuritis, and *pneumonia* may be simulated or really present, being caused by the extension upwards of the suppurative process through the right side of the diaphragm; but a careful investigation of the history of the case will usually elucidate the problem. The nature of the pus withdrawn or expectorated is usually conclusive in amœbic cases.

Hydatids are not likely to be mistaken for abscesses owing to their slow and painless growth and to the clear evidence of fluctuation; but when suppuration occurs in the cyst the diagnosis may be impossible without the use of the aspirator needle. The presence of hooklets will settle the diagnosis in most cases.

Pyonephrosis will be differentiated by the small range of mobility of the tumour in respiration, by the evidence of the colon being in front of it, and often by the periodical discharge of large quantities of pus in the urine.

Abscesses in the abdominal wall, sub-phrenic and peri-nephritic abscesses can only be eliminated in the diagnosis

by a careful investigation of the history in each case and by microscopic examination of a sample of the pus removed by aspiration.

Prognosis.—The outlook is serious in all cases of an acute nature, but favourable in the chronic ones. About half of the patients affected with solitary abscesses recover. Expectoration of the pus is sometimes followed by complete recovery, but the possibility of operative interference is the main feature in a favourable prognosis.

Treatment.—Where the situation of the abscess permits of its being reached by a *free incision*, this should be performed without delay. Usually it will be found advisable to suture the liver to the abdominal wall, and after adhesions have formed the abscess may be opened by the scalpel or thermo-cautery. This method gives good results in the amœbic abscess. Free drainage must be provided, and Lafleur advises the washing-out of the abscess-cavity with quinine solution.

Aspiration of the cavity in chronic tropical abscess is often successful without incision, but it is not without grave danger. Manson's operation is considered by many surgeons practising in the tropics to be satisfactory. A large trocar and cannula are pushed into the abscess, and upon withdrawal of the trocar a full-sized rubber tube is inserted deeply through the cannula, and the contents are slowly syphoned off. It may be necessary in some cases to fasten the shield of the cannula to the skin in order to maintain it in position for some days, after which it may be drawn over the rubber tube, which can be permitted to remain *in situ* till contraction of the cavity warrants its removal. Maitland condemns this method and other plans of puncture, and advises that the abscess should be opened through the pleura after resection of a rib, or through the abdominal wall after the peritoneal surfaces have been stitched together. Abscesses of the lung and pleura may also be treated by free incision, and, if necessary, by resection of one or more ribs.

Prophylactic measures are of primary importance in the case of Europeans resident in tropical or sub-tropical regions, the free use of alcohol and over-indulgence in animal food and condiments being answerable for a large percentage of tropical abscesses independent of the incidence of dysentery.

Drugs are of little value, but the early stage of tropical hepatitis before pus has formed is believed to be modified by large doses of ammonium-chloride.

LIVER, ACTINOMYCOSIS OF

Under 'Actinomycosis' the general symptoms of the disease caused by the ray-fungus have been described. The liver, when infected, is nearly always invaded in a secondary manner.

The symptoms and signs are obscure, but the clinical and pathological picture is one of solitary or multiple abscesses containing foul-smelling pus, which often finds its way between the numerous adhesions which connect the liver with coils of bowel and the abdominal parietes. Death from peritonitis or pyæmia is the almost invariable rule. Auvray reports thirty-one cases in medical literature, six of which had been submitted to surgical operation; but all ended fatally after a long interval. Mayo Robson has, however, successfully removed the growth from the gall-bladder and liver in one case, proving that the only hope of recovery lies in early and complete extirpation, followed by large doses of iodide of potassium.

LIVER, ACUTE YELLOW ATROPHY OF

This rare disease, also known as Malignant Jaundice or Icterus Gravis, is an acute toxæmia manifesting itself by cerebral symptoms, jaundice, and hæmorrhages, and eventuating in a shrinking of the liver-dimensions caused by rapid degeneration of the hepatic cells.

Etiology.—It is more common in women than in men—a fact probably explained by its association with pregnancy in some instances. The only case seen by the writer was a typical one, occurring about the sixth month of pregnancy. Less than 300 cases in all are found recorded in medical literature by Hunter. It may occur at any age, but it is extremely rare in childhood; the majority of the recorded cases occur between the ages of twenty and thirty.

There is no evidence of any specific etiological factor, though many such have been suspected or alleged, if we exclude its undoubted association with the pregnant condition. Cases have been reported as occurring during or after certain infectious diseases or as following severe frights or profound emotional states, but the majority of recorded instances are in persons who were in a healthy state up to the time of the advent of the jaundice. In a few examples of the disease there was evidence of previous cirrhosis or obstructive jaundice.

There can, however, be little doubt that the disease is the result of some virulent poison which causes rapid degeneration

not only of the hepatic cells, but of the kidney, heart, and other organs. Whether this poison is introduced from without or manufactured within the organism is a subject for future demonstration. The close resemblance between the results of phosphorus-poisoning and acute yellow atrophy has led some authorities to regard the pathological process as identical, and Kobert goes so far as to affirm that the disease is a toxæmia caused by phosphoretted hydrogen generated by bacterial action in the intestine from the phosphates normally present.

Hitherto no micro-organism has been demonstrated in constant association with the disease, though its occasional occurrence during epidemics of infective jaundice (also termed 'icterus gravis') lends some support to the view that acute atrophy may be the result of an infective microbe, and some authorities continue to regard the disease under consideration as but a severe endemic variety of infective jaundice.

Pathological Anatomy.—The most obvious feature is the marked diminution in the mass of the liver, which may amount to only half its normal bulk and weight. The organ is of a yellow colour, which is very intense in patches; its surface is smooth and its capsule wrinkled; the hepatic tissue is soft, and the liver may be found folded upon itself and lying close to the spine. On section the microscope reveals extensive destruction of the hepatic cells, those which still remain being filled with fatty granules and having their contours faint or indistinct, especially towards the periphery of the lobule where the degeneration commences.

Around the yellow patches of degenerated tissue are seen areas of red and firmer substance consisting mostly of fibrous stroma from which the cells have entirely disappeared, only fatty granules and detritus, with crystals of tyrosin and leucin, being visible. These red patches or areas cut with some difficulty owing to the preponderance of fibrous tissue, they represent the advanced stage of the degeneration, and contrast in consistency with the yellow softened masses which they surround. The small bile-passages are usually blocked with secretion and desquamated epithelium; they may finally disappear as the neighbouring hepatic cells perish. The larger ducts and the gall-bladder are generally devoid of bile.

In some instances evidence of attempts at repair have been observed in the proliferation of the hepatic cells which had escaped in the rapid granular or fatty degeneration.

The kidney-cells, the epithelial lining of the stomach, intestines, and bronchi, and the muscular fibres of the heart show similar degenerative changes, proving that the disease is the result of a universal or widespread toxæmia in which the liver-cells suffer along with those of nearly every tissue

in the body. There are always, in addition to the pathological changes above mentioned, the deep bile-staining of all the tissues and the evidence of hæmorrhages in various localities.

Symptoms.—The onset of the jaundice is usually preceded by gastric disturbance, as in the ordinary catarrhal form, but a mild degree of fever is usually present. After the staining of the skin becomes obvious the anorexia, malaise, and occasional nausea or vomiting may continue for a week or ten days, when, with more or less abruptness, the serious and characteristic nervous symptoms are ushered in with restlessness, drowsiness, stupor, delirium, severe vomiting, and sometimes maniacal excitement.

In a few hours the case passes from that of an ordinary catarrhal jaundice to the grave aspect of a deep typhoid condition, in which the jaundice rapidly intensifies, and the vomit becomes black from the ejection of altered blood and bile. Convulsions supervene in a majority of the cases, and death usually follows soon after the advent of profound coma.

The liver-dulness is found diminished soon after the case enters upon its second or acute stage, and the organ may be found on percussion to continue to shrink from day to day in thin subjects, where physical examination is most easily carried out. As it doubles upon itself and falls backwards from the abdominal parietes all traces of liver-dulness disappear in those patients who linger for several days. More or less pain is complained of in the hepatic region before consciousness is blurred or dissipated. Palpation will usually show some enlargement of the spleen ; hæmorrhages under the skin and from the mucous surfaces are commonly present.

At this stage the temperature is normal or sub-normal, but the range varies greatly ; often it rises just before death, but it may continue below the normal from the first onset of malaise till death.

The urine exhibits marked changes. From the first it is bile-stained owing to the presence of the ordinary pigments, and some albumin and casts are generally to be found ; but its most distinctive character is the presence of leucin, tyrosin, and other products indicative of perverted metabolic action. The former substance occurs in spheroidal striated masses of minute crystals on evaporating the urine ; tyrosin is found in needle-shaped crystals arranged in the form of sheaves, fans, or discs. The output of urea is greatly diminished and may fall to nothing, though the excretion of nitrogen in the form of uric acid and ammonia is much augmented. Leucin and tyrosin have been found in the liver even when absent from the urine, so that their production, though not absolutely characteristic, is strongly suggestive of the disease ; sarcolactic acid is also generally present. Acute yellow atrophy generally proves fatal within a fortnight, but cases have been reported

with a duration of several weeks. When occurring during pregnancy the progress is generally very acute; in the case seen by the writer death occurred inside a week. As a rule, after the onset of the second stage the duration of the cerebral symptoms is only a matter of two or three days.

Diagnosis.—*Phosphorus-poisoning* can be recognised by the history of the poison having been swallowed; by the more marked symptoms of gastritis at the very beginning; by the *enlargement* of the tender liver, and by the jaundice being less intense. Though tyrosin and leucin have been both found in the urine in acute phosphorus-poisoning, the latter, if at all present, exists in the merest traces, and the tyrosin never in the quantities usually seen in acute atrophy. The deceptive lull between the disappearance of the gastritis which follows immediately after swallowing the poison and the onset of the jaundice two or three days later is characteristic; if the patient survive long enough the liver may diminish somewhat in bulk.

Jaundice, when severe and following other causes than atrophy, as in hypertrophic cirrhosis, gall-stones, cholangitis, &c., cannot be readily mistaken if the increased size of the liver be noticed. The depth of the cerebral symptoms, with the diminution in hepatic dulness and the discovery of tyrosin and leucin, may be taken as conclusive in all doubtful cases of severe jaundice.

Prognosis.—This is always grave; though cases ending in recovery have been recorded there is still always suspicion of an error having been made in diagnosis. Some examples of the disease do run on for many weeks, and it is in such that recovery has been reported. Pregnancy certainly deepens the gravity of the outlook, death being very likely to follow rapidly after abortion.

Treatment.—Nothing can be done save upon the lines of relieving the most urgent symptoms, as vomiting, hæmorrhages, &c. Calomel, purgatives, and warm saline intravenous injections may be tried with some hope of prolonging life for a short time.

LIVER, ATROPHY OF (CHRONIC)

Simple atrophy of the liver, in which the organ diminishes uniformly in size independent of cirrhotic and degenerative changes in the parenchyma, is mainly, if not entirely, confined to conditions caused by inanition, marasmus, and senile decay.

The hepatic atrophy produces no special symptoms, and as a rule is only occasionally recognisable when the volume of the organ shrinks so considerably as to be capable of markedly diminishing the extent of the normal hepatic dulness. The primary causes, as stricture of the gullet,

pyloric obstruction, anorexia nervosa, intestinal chronic catarrh, &c., produce each its own characteristic clinical picture, which usually entirely overshadows the liver-atrophy, till the *post-mortem* examination reveals the diminished bulk of the organ. The lobules are then found to be diminished in size, the hepatic cells are smaller than the normal, and in many places may be so wasted as to be unrecognisable, their location being only marked by the remnants of the capillary network which originally surrounded them. The liver is firmer in consistency, though there is no absolute increase in its fibrous tissue, and it may scale less than half the average weight.

The prognosis and treatment of the condition are those of the primary diseases which have led to the hepatic wasting.

LIVER, AMYLOID DISEASE OF

Under 'Lardaceous Disease' on page 770, the etiology and general characters of this form of degeneration are described; it follows prolonged suppuration from whatever cause, or appears in association with syphilis. The disease is never confined to a single organ, but invades liver, spleen, kidney, and intestines.

Symptoms and Signs.—As a rule, there are no symptoms till other organs become involved, and diarrhœa from amyloid degeneration of the intestine may first cause attention to be directed to the abdominal contents. Kidney-involvement may in a similar manner be the only indication of the affection which has already seriously infiltrated the hepatic tissue. The explanation of this lies in the fact that the degeneration does not interfere appreciably with the functions of the liver till the end. There is no jaundice nor ascites; but in the last stages of the affection, when the pressure of the amyloid deposit in the walls of the small blood-vessels has caused atrophy of the hepatic cells, there may be diminution in the amount of bile secreted, the stools become pale, the urine shows less pigment, and there is a fall in the amount of urea excreted.

The characteristics of the amyloid liver, as appreciated during life, are a uniform painless enlargement which may reach to the extent of three or four times the normal bulk of the organ. (Thus, instead of the liver weighing one-fortieth part of the body, it may scale as high as one-tenth of the entire body-weight.) Its surface is uniformly smooth and its margins sharp or occasionally somewhat rounded; to the fingers it is firm and resistant, though elastic, and no tenderness is elicited by pressure. The vertical hepatic dulness in

the mammillary line may be increased from four to eight or nine inches.

Splenic enlargement is usually well marked, and the firm consistency of the tumour is obvious. The urinary examination reveals albumin, much increase in the volume of the excretion, with low s.g. and some hyaline casts. When the intestinal surface is involved, obstinate diarrhœa is present.

Pathological Anatomy.—The liver will exhibit the characters which have been obvious on clinical examination. Its colour is pale, resembling a piece of phosphorus or translucent wax; the appearances have been also likened to boiled bacon or raw ham. The microscope reveals extensive infiltration of waxy or lardaceous deposit around the coats of the capillaries, especially those of the middle zone of the lobules. The cells of the liver-parenchyma are atrophied by pressure, but only occasionally lardaceous degeneration may be demonstrated in their protoplasm. The characteristic mahogany-brown discoloration produced by washing the cut surface of the organ in a watery solution of iodine and potassium iodide is readily obtained.

Similar changes will be found in the spleen, kidney, and intestines.

Diagnosis.—The above signs of liver-enlargement without jaundice supervening upon chronic suppuration from any cause, as in bone-disease, phthisis, &c., or in association with syphilis, and less frequently with the cachexia following cancer, are characteristic, especially when there is a splenic tumour without ascites. The urinary characters are of first importance in doubtful cases.

Prognosis.—As the functions of the liver are so little interfered with, the gravity of the prognosis lies in the direction of the extent of the degeneration in other organs, notably in the intestine and kidney. There is no doubt that moderate degrees of amyloid infiltration of the liver entirely disappear when the cause of the prolonged suppuration can be removed by surgical procedures.

Treatment.—Beyond the imperative necessity of removing the cause of the primary disease, when this is possible, little else can or need be done. Iodides are believed to possess considerable power over the progress of the degeneration when surgical interference is impossible, and they are especially indicated in syphilitic cases.

Prophylactic treatment is of great importance, and the early removal of all suppurative lesions constitutes a reliable safeguard in every case. Even in pulmonary phthisis much may be done by the judicious use of agents which tend to check the suppurative process in the lung-cavities by suitable pure open-air treatment, creosote, and other measures.

LIVER, CANCER OF

Primary carcinoma of the liver is comparatively rare; the great bulk of all cases afford unmistakable evidence of the disease being secondary to malignant disease in other organs. Occasionally, however, a primary hepatic cancer may give rise to secondary deposits in the gall-bladder or ducts, and it may be difficult during life, or after death, to decide which is the primary focus.

Etiology.—Heredity in hepatic cancer, independent of the general hereditary influence seen in the production of carcinoma, appears to be an etiological factor in a moderate percentage of cases.

The primary disease is more frequent in men and the secondary in women, since the breast, uterus, and appendages are so frequently the seat of the primary affection.

Age, as in carcinoma of other organs, exhibits its influence, as the secondary disease is comparatively rare before forty, and most of the cases occur between the ages of forty-five and sixty-five. Primary cancer of the liver is not infrequently met with in young children.

Trauma seems to be an etiological factor sometimes, and probably it operates more frequently in the primary juvenile form.

Allied to trauma is the influence of irritation, seen in the frequency with which the presence of gall-stones is associated with cancerous formations in the gall-bladder, liver, and bile-passages. Most of the cases of carcinoma of the gall-bladder associated with gall-stones are examples of *primary* cancer occurring late in life; the disease is at least four times more frequent in women than in men, whilst primary disease of the liver itself is more common in males.

Pathological Anatomy.—Primary hepatic cancer is met with in three different forms—viz. (1) *massive*; (2) *nodular*; (3) *infiltrating*, or *cancer with cirrhosis*. In the first mentioned a large mass of firm carcinomatous tissue is found anywhere in the interior of the organ surrounded by healthy hepatic tissue. In the second variety the liver is throughout studded with nodules of a yellowish-grey colour, the condition presenting the appearance seen commonly in secondary cancer. In the cirrhotic or infiltrating form the liver may be little enlarged, and the enlargement is uniform. The surface is uneven, with nodular projections or flattened ridges visible beneath its capsule. This type is regarded as an adeno-carcinoma; there is considerable proliferation of the connective tissue, with diffuse carcinomatous affection of the hepatic parenchyma. The massive type of hepatic cancer consists usually of spheroidal-celled carcinoma of rapid growth; the

nodular has similar histological characters, but sometimes the cells are columnar in both, or show transitional forms. In the cirrhotic variety there are areas where the microscopical characters point to a simple trabecular adenoma, but as these progress they develop into typical alveolar carcinoma, or they may remain tubular. Some authorities refuse to regard the form of adenomata associated with cirrhosis of the liver to be of a cancerous nature, as they interpret the new-growths in this variety as exaggerations of the hobnails caused by an attempt at compensatory hypertrophy of the hepatic cells. But there can be little doubt that the adenomatous cirrhosis tends ultimately to pass into a true carcinomatous cirrhosis, in which the growths are liable to invade the portal and hepatic veins, causing thrombosis.

Secondary cancer of the liver is produced by the morbid material being conveyed by the hepatic artery as in mammary cancer, or by means of the portal vein as in carcinoma of the stomach, intestines, uterus, and pancreas. These metastatic growths may increase so rapidly as to overshadow the primary focus. They are nearly always multiple, forming large nodules which usually project from the surface of the liver; occasionally secondary cancer may take the form of a general infiltration of the organ. The nodules are white or yellowish-white, and exhibit a marked tendency to degenerate, so that by cicatricial contraction often their summits are cupped or umbilicated in the centres. The deposit may increase to the extent of destroying all but the remnants of the hepatic parenchyma, and the affected liver may weigh, in rare cases, as much as thirty pounds, or even more. Owing to pressure on the bile-ducts, the organ is often deeply stained by retained bile; and when the secondary deposits invade the region of the transverse fissure, jaundice and ascites may both be present. The deposits may be scattered evenly throughout, but usually they are much larger in one lobe than in the other, and they often project from the edge or lower surface, which is usually very irregular in outline. Secondary changes may be observed, by which the contents of some of the nodular masses show cystic degeneration, especially in the squamous-celled variety, and colloid carcinoma is not rare. Spheroidal-celled structure is commonly found in the rapidly growing nodules, and intermediate forms between this and the columnar-celled are frequent. The white masses may be hæmorrhagic in the centre, and pus may even form, whilst around their peripheries more or less cirrhotic or sclerotic change is also present owing to the irritation of the connective-tissue elements. Where the disease has commenced as a primary carcinoma of the liver or of the gall-bladder the organ may become studded throughout with secondary nodules.

Symptoms.—The cancerous cachexia, with its mixed features of anæmia and emaciation, often arrests the patient's attention before he is aware of any local lesion. Then follow loss of appetite, progressive weakness, and pain, felt first in the right hypochondrium or epigastrium, and afterwards radiating towards the shoulder, over the upper zone of the abdomen, or downwards towards the groins ; occasionally pain is entirely absent. Jaundice appears in about half the cases, and is much more common in secondary hepatic cancer ; and the same remarks apply to the frequency of ascites, both conditions being most commonly the result of secondary nodules in the glands or tissues about the hilum. Œdema of the legs may be extensive when the growths invade the vena cava as well as the portal vein. An important feature in the jaundice of hepatic cancer is its persistence and progressive character.

Though rapid and progressive emaciation is invariably present, the gross body-weight may increase owing to the enormous growth of a rapid primary cancer, and it should never be forgotten that a similar result may follow in secondary cases from the accumulation of ascitic fluid, so as to lead to a serious error in diagnosis. Fever is sometimes present, and intensifies the amount of emaciation, which is usually more rapid in secondary as contrasted with primary hepatic cancer ; death is usually the result of asthenia, but often coma, following upon delirium, supervenes.

The physical signs in the liver are usually well marked. Often upon *inspection* the organ is obviously enlarged, causing bulging or distension of the upper part of the abdomen ; and where the abdominal walls are thin the larger nodular masses may be visible during quiet breathing, and when situated near the margin they may be seen to move upwards and downwards on deep respiration. The superficial veins may be distended.

Upon *palpation*, the edge of the enlarged organ may be felt below the umbilicus. It is very hard and irregular from the presence of nodular growths ; sometimes in secondary cancer these may be detected to be cup-shaped or umbilicated at their summits. Nodules may often be detected about the navel, or palpated in the region of the falciform ligament. The organ may be markedly tender upon pressure, and a friction-fremitus may be detected on deep inspiration, which brings superficial nodosities under the hand applied flatly to the abdominal wall.

Percussion will corroborate the signs obtainable by inspection and palpation. Sometimes the organ may be found uniformly enlarged, but careful examination will nearly always reveal want of symmetry in the enlargement, the right lobe usually showing more swelling than the left, even when nodosities are entirely absent. In one case observed by the

writer the smooth anterior surface and sharp depressed margin of the liver were felt throughout the progress of the disease as quite normal, but the hepatic vertical dulness in the mammillary and right axillary lines was of more than double the normal limits. The autopsy revealed an enormous mass of firm carcinomatous tissue buried in the centre of the right lobe, leaving the entire hepatic surface normal in appearance. It is not rare in some cases to find a nodular mass appear somewhere on the liver-surface or margin within two or three days after the last negative examination.

The vertical dulness in the right mammillary line may sometimes be found increased from four to ten inches or more, and often in rapidly progressive cases the increase may be appreciable upon percussing the organ every four or five days.

The spleen is rarely enlarged, but the gall-bladder may often be found hard and distended, and occasionally right pleural effusion results from the extension upwards of the carcinomatous infiltration. In the infiltrating form of primary hepatic cancer there may be no appreciable enlargement of the organ, which in rare cases may be even atrophied.

The physical examination in secondary hepatic carcinoma will often reveal the site of the primary affection in some other organ, and this so often overshadows the liver-involvement that, in perhaps half of the cases, the latter is not suspected or evident. Upon the other hand, the primary focus may remain latent, the secondary disease of the liver, by the rapidity and massive proportions of its growth, entirely overshadowing it.

Sarcoma of the liver is much rarer than carcinoma, being in its primary form one of the rarest of diseases, and an affection which cannot, during life, be differentiated from hepatic cancer. The secondary form of sarcoma is often latent, but sometimes the growths assume large proportions, either in the form of a general infiltration or as extensive nodular masses, which are rarely or ever cupped in the centre, and which are very liable to hæmorrhagic extravasations.

The characters of the hepatic deposits are of the same type as the primary growth which originates in the bones, the choroid, or in the skin or other regions. The condition is recognised only after a careful search for the original focus, which may not infrequently be found to be a melano-sarcoma, starting in pigmented nævi in the skin ; and other metastatic growths may usually be found in different parts of the body.

The state of the urine (melanuria) in melanotic cancer is often distinctive.

(*Adenomatous* growths of the liver of an innocent type, *simple cysts*, and *cavernous angeiomata*, are occasionally met with, but as a rule they produce no clinical symptoms.

Tubercular growths are always, when present, part of a general infection, as are *lymphadenomata* of Hodgkin's disease.)

Diagnosis.—The early stage of *hepatic cirrhosis* may be mistaken for malignant disease, but with care it can be differentiated in the great bulk of cases by attention to the following points. The enlargement is always uniform, and in the early stages smooth; it is of slow and chronic course, and when at a later stage nodosities may be felt these are invariably small and never cupped in their summits. Some tenderness may be present, but generally it is much less than that met with in cancer. There is no marked cachexia, and the jaundice differs from that seen in carcinoma in never being so intensely green in hue; moreover, the history can usually be obtained that the jaundice has come and gone or shown obvious degrees of staining at different times, whilst in cancer its steadily progressive and permanent character is notable. Even when the skin is deeply yellow in cirrhosis there is always some bile in the motions. The spleen is often enlarged, the gall-bladder seldom distended, and hæmorrhoids are usually present—opposite conditions to those met with in cancer. The patient's age may be well below forty, and the history of alcoholism is usually obtainable. Life may be prolonged for many months after the onset of jaundice and ascites, whilst the advent of these in cancer usually precede death by only two or three months. After the removal of ascitic fluid in the one case large nodules in an enlarged organ may be felt, but in cirrhosis the small hobnails are found in a liver of diminished size.

When cirrhosis and cancer exist together the diagnosis is most difficult, as the liver may not show marked enlargement; a correct judgment can only be arrived at by a careful history of the case and a patient observation of its progress. The frequency of splenic enlargement in simple cirrhosis is a noteworthy feature.

Fatty and *amyloid degenerations*, with the history and uniform painless and smooth enlargement unassociated with wasting, ascites, or jaundice, are easily eliminated, but when a large syphilitic gumma complicates amyloid disease the diagnosis is difficult, and can only be certainly made by the discovery of other syphilitic lesions and by the marked effect of iodide treatment—points which will also serve to establish the presence of *syphilitic disease* of the liver independent of amyloid disease.

Hydatid cysts, especially of the multilocular or alveolar type, are differentiated by their chronic history and absence of cachexia and by the presence of splenic enlargement. The simple hydatid cyst is not liable to be mistaken for cancer, owing to its smooth surface, elastic feel, fluctuation, slow growth, and painlessness.

Gall-bladder tumours caused by the presence of calculi, which have produced inflammatory thickening and swelling, may be mistaken for carcinoma of the gall-bladder or of the liver. The absence of cachexia and the presence of a history of gall-stones, with the length of the duration of the case, are usually sufficient for differentiation, but the high percentage of cases in which a true primary cancer of the gall-bladder follows upon the lengthened irritation caused by gall-stones should always be borne in mind. (See under 'Gall-bladder Cancer,' p. 429.)

Sarcoma of the liver, as already stated, is almost always secondary to similar growths in other regions, and the discovery of primary or secondary sarcomatous growths in the skin, choroid, or elsewhere, with black urine, will point to the nature of the massive secondary growth in the liver being a melano-sarcoma.

Enlargement of the liver caused by the passive congestion of *mitral disease* may be extensive and associated with tenderness, ascites, and jaundice; the presence of the valvular lesion, the slow progress and history of the case, and the fact that the hepatic swelling varies greatly from time to time, whilst it always remains symmetrical, are distinctive clinical features.

Prognosis.—This is invariably fatal. In primary cases death may occur in less than four months; taking all cases together, the duration is usually well under one year. Every week that a patient lives beyond the twelve months following the onset of a suspicious hepatic tumour justifies a serious doubt in the diagnosis. Where cancer has been reported as found in the liver two years after the onset of symptoms of hepatic trouble, it may be safely assumed that carcinoma supervened upon some other liver-condition at a period much later than the onset of the original symptoms.

Treatment.—Symptoms like pain, vomiting, &c., should be treated by appropriate remedies. In carefully selected cases a considerable amount of success has followed surgical procedures when the growth was circumscribed or confined to the region of the gall-bladder. Anchütz's recent list of ninety-six operations on the liver performed for neoplasms shows that only seventeen died from the operation itself.

LIVER, CIRRHOSIS OF

This term, first applied by Laennec on account of the tawny yellow colour exhibited by the diseased liver, is now employed to designate several diseases of the organ, in which the main pathological feature is an increase of fibrous tissue, the result of chronic inflammation. These separate affections

have of late years been variously classified by authorities on pathological, etiological, and clinical bases. The result of such attempts has led to not a little confusion from the overlapping of the different symptoms and pathological processes. The subject will be simplified by first describing the most common form of the disease.

ATROPHIC CIRRHOSIS, INTERSTITIAL CHRONIC HEPATITIS, LAENNEC'S CIRRHOSIS, or ALCOHOLIC CIRRHOSIS

Etiology. — Alcohol is the cause in almost every instance of the disease. In proportion to the percentage of alcohol in the beverage, the incidence of hepatic cirrhosis seems to increase. Thus it is rare in beer-drinkers and in countries where light wines are consumed; but where the usual beverage is distilled spirits, either in the form of whisky, gin, or brandy, it is comparatively common, especially amongst those who drink strong spirits before food. In Britain it would appear as if the disease is somewhat less frequently met with of late years, since the outcome of every small increase in the taxation of spirits has resulted in the public being supplied with a more diluted alcohol. The important question arises whether alcohol is the sole agent in the production of the disease, or if the interstitial inflammation may not be the result of some of the other products of fermentation contained in the alcoholic beverage. The writer has satisfied himself that atrophic cirrhosis is more prone to occur in those who drink the purer spirit which is the product of the patent still; but this result may be caused by the consumers of the silent spirit indulging in a more concentrated form of beverage.

The disease is much more frequent in males. Middle-life is usually the period of its onset; but it has been reported sometimes as occurring in children, and some authorities have recorded cases where apparently the disease has been congenital in the children of drunkards.

Syphilis appears to be an etiological factor in some instances, which may explain the congenital cases; and it can hardly be doubted that it increases the incidence of cirrhosis amongst those who drink concentrated spirits. In a similar manner, the disease is met with more frequently in intemperate subjects who have suffered from infectious diseases, diabetes, malaria, valvular lesions, phthisis, &c. Individual peculiarity plays an important *rôle* in the production of cirrhosis, since many drunkards escape the manifestations of the disease whilst others succumb to small amounts of the poison. Adami maintained that the cirrhotic change may be the result of an irritation produced by a small diplococcus, which he believed to be a modification of the colon bacillus; whether the presence of this micro-organism is the cause or the result of the disease

remains to be proven. He found the microbe in small numbers in livers which were not cirrhotic.

Morbid Anatomy.—In the early stages of the disease the organ may be enlarged, but opportunity of *post-mortem* examination is not sufficiently frequent to determine this as an *essential* feature, though clinical evidence is strong upon the point. When the cirrhotic liver is found enlarged after death, the increase is found to be due mainly to the presence of fat, and not to fibroid proliferation alone.

The atrophic liver is characteristic. It is considerably reduced in size and may weigh only one-third of the normal. Its surface is studded over with small protuberances, the largest of which seldom exceed the size of a sloe. The resistance of the organ is greatly increased, being firm and leathery on section, which exhibits rounded patches of tawny brown or greenish-yellow colour separated from each other by light-coloured bands of fibrous tissue. Each patch or island of hepatic tissue is found under the microscope to consist of the remnants of one or more hepatic lobules. The surrounding greyish-white tissue consists of white fibres with more or less yellow elastic strands intermingled with it. The fibrous growth passes between the lobules and into each, following the distribution of the capillaries. The hepatic cells are atrophied, bile-stained, and often much infiltrated with fat. The walls of the blood-vessels arising from the branching of the portal vein, which pass between the lobules, are thickened, and more or less obliteration results. The capillaries of the lobules being entirely destroyed in many places explains the portal stasis, which, with the increase in the fibrous tissue, constitutes the main features of the disease; the fibroid proliferation, by its pressure and subsequent shrinkage, causing the ruin of the hepatic cells and obliteration of the bile-ducts. In the fibrous tissue are found many new vessels derived from hypertrophic changes in the capillaries of the hepatic artery, and alongside the destructive processes attempts at regeneration of the hepatic cells are visible in the formation of adenomatous nodules.

Occasionally the atrophied cirrhotic liver may be smooth, or almost entirely devoid of nodular protuberances or superficial granulations, but its fibrous tissue is nevertheless greatly increased, and its microscopical characters identical with the hobnailed variety.

The shrinkage in the size of the organ is the result of the tendency of the new fibrous tissue to contract; hence it is an essentially slow process. In those cases where the cirrhotic liver weighs several times its normal avoirdupois, the surface is usually smooth or but faintly granular; and it is probable that, had life been prolonged sufficiently, many such smooth organs would have presented the usual hobnailed character.

Osler describes this form of the disease under the title of the 'fatty cirrhotic liver,' which, he states, occurs in beer-drinkers.

Other pathological changes are observed in atrophic cirrhosis, as splenic enlargement, renal cirrhosis, interstitial pancreatitis, and not infrequently disseminated tuberculosis.

Symptoms.—For years no symptoms may be observed; often marked atrophic cirrhosis is found after death where no suspicion of such a condition had been formed during life. This result probably depends upon the extent to which the collateral circulation becomes established, as will be referred to later on.

In the most typical cases of the disease there are first seen recurrent attacks of hepatic congestion, in which the liver is found to be enlarged, somewhat tender, and firm to the touch. Such attacks often are observed to follow upon bouts of unusually severe indulgence during the course of chronic alcoholism, and often a slight but unmistakable degree of transient jaundice accompanies each. After a more or less variable period, determined by the habits of the individual, the liver may be found to remain constantly enlarged, and often tender. In this condition it may remain to the end, or after many months shrinkage may set in. The prominences or hobnails on the surface of the enlarged organ will be evident on palpation if its lower edge projects below the costal arch.

The term Hypertrophic Cirrhosis is, unfortunately, often loosely applied to this stage of cirrhotic enlargement from alcohol. The title should be reserved for another type of the disease, first described by Hanot, which will be dealt with in the succeeding article. It is highly probable that most, if not all, cases of atrophic alcoholic cirrhosis pass through this preliminary stage of enlargement, if opportunity for careful examination has been afforded from the beginning.

The symptoms which usually cause the patient to first seek advice are those produced by portal obstruction. The stasis of the blood in the portal system usually manifests itself in early gastric derangement; there is loss of appetite, furred tongue, abiding nausea, and often constant retching or vomiting, beginning as soon as the patient awakes in the early morning or attempts the first meal of the day—symptoms which are identical with those produced by the temporary gastric catarrh of an earlier stage. Hæmatemesis may appear suddenly and be very profuse. It results either from the passive congestion of the gastric mucosa, in which case it is usually slight, but more frequently it is the result of rupture of large varicose veins which are situated at the lower end of the œsophageal opening into the stomach. When from this latter source, the hæmorrhage may be very alarming:

enormous quantities of black liquid blood, resembling blackberry-juice, slowly but continuously well up into the mouth, till the patient succumbs from syncope. Hæmorrhage from the bowel is also common, and it is not unusual for the hæmatemesis and melæna to be the first symptoms to attract attention. Less often epistaxis is troublesome at this early stage, though it is frequently present later on, when, owing to changes in the blood itself, hæmorrhages are frequent into the skin, pleural cavity, and retina, or from the mucous membrane of the genito-urinary tract and other regions.

The spleen is usually enlarged at an early stage, and as the portal stasis increases the organ may be easily felt. The intestinal congestion may reveal itself in diarrhœa and the development of large hæmorrhoidal growths.

Ascites occurs gradually, and the abdomen becomes distended so insidiously that the patient frequently imagines that the enlargement is due to obesity ; occasionally the effusion is sudden. The appearance of ascites is generally a late symptom, but often a very considerable degree of effusion exists when the liver is still markedly enlarged, and by suddenly ' dipping ' the digits at right-angles to the surface of the abdominal parietes the layer of liquid is displaced, and the enlarged and firm organ is brought into contact with the finger-tips. Some œdema of the lower extremities may precede the ascites, but more frequently it follows the abdominal distension.

The veins on the surface of the belly increase greatly in calibre and prominence, owing to attempts at the establishment of a collateral circulation. This is effected by a portion of the portal blood reaching the right auricle without permeating the liver, which it may do by the following communications or routes : (1) The blood in the inferior mesenteric and superior hæmorrhoidal veins, which are radicles of the portal, may through the hæmorrhoidal plexus pass into the general venous system, by means of the iliac veins; (2) the inferior œsophageal communicates at the opening of the diaphragm with the gastric veins conveying the blood to the azygos ; (3) the para-umbilical vein of Luschka, running in the ligamentum teres (derived from the portal through an unoccluded portion of the umbilical vein), joins with a branch of the deep epigastric vein, thus establishing a communication between the portal and general venous systems through the external iliac ; (4) Sappey's accessory portal veins, which run between the reflections of the peritoneum, forming the ligaments of the liver : these connect the portal with the general venous system through the phrenic veins ; (5) the retro-peritoneal veins sometimes become considerably enlarged and capable of diverting much blood into the general venous system from the portal radicles without its flowing through the

liver; (6) it is not uncommon for new vessels to form in adhesions between the liver and diaphragm.

When the enlargement of the para-umbilical vein in the round ligament is marked, there is sometimes a large cluster of dilated veins round the umbilicus where this vessel empties itself into the upper epigastric veins. To this protuberant mass of varices the title of 'caput Medusæ' has been given, and occasionally a venous murmur may be audible over it.

The effusion into the peritoneal cavity may reach great dimensions; as much as four or five gallons have been removed in some cases (see under 'Ascites'). The fluid is albuminous, clear, and yellowish, or of a deep amber, and with a s.g. of 1012 to 1015. When chronic peritonitis has supervened, it may contain shreds of lymph and some blood, and may coagulate with great rapidity after its removal from the body.

Effusion into the right pleural cavity is not rare. When the collateral circulation above described becomes well developed, ascites in some cases may not supervene; or it may remain stationary without increasing; or it may even be seen to diminish—a rare condition, in which hæmorrhages are very liable to occur.

Jaundice is not a usual clinical feature of alcoholic cirrhosis, but a slight icteroid tinge of the skin and conjunctiva is always present; occasionally, as already mentioned, transient attacks of jaundice supervene, especially after bouts of unusual intemperance. The urine is scanty, of high s.g., deposits urates on cooling, is of a reddish colour from the presence of urobilin, but seldom contains ordinary bile-pigment. The total output of urea is diminished considerably owing to the interference with metabolism, but the ammonia is increased. Albuminuria at an early stage indicates concurrent renal disease, but traces of albumin may appear, as the abdominal pressure causes some renal congestion.

Fever is usually absent throughout; occasionally, however, a sub-febrile temperature may be recorded. Emaciation soon shows itself after the appearance of symptoms of portal obstruction; as this progresses, the clinical picture of cirrhosis becomes unmistakable. The wasting of the legs, when these are not œdematous, contrasts strongly with the pot-bellied condition; the face becomes pinched, the malar prominences more marked; venous stigmata show obviously upon the sallow, wasted cheeks; the breathing becomes quick on slight exertion, and an aspect of dejection, often heightened by drowsiness or stupor, supervenes, which is characteristic.

The supervention of general tuberculosis, or of tubercular peritonitis, may cause corresponding changes in the symptoms, and always hastens the final issue.

Toxæmia not markedly differing in character from that seen in renal disease is often witnessed in the later stages, when the kidneys may be intact. The patient may sink into a comatose state, after a stage of delirium, or he may succumb to an inveterate diarrhœa, or suddenly pass away from the shock of a copious hæmorrhage which ends in a fatal syncope precipitated by cardiac degeneration.

The duration of the disease is variable, and cannot be even approximately determined, owing to its insidious commencement; but it has perhaps always a course of several years. After the appearance of obvious signs of portal obstruction, the duration of the patient's life will, in the great majority of cases, rarely exceed a limit of several months.

Diagnosis.—The presence of ascites in an alcoholic patient whose spleen is known to be enlarged and who has suffered from hæmatemesis or melæna, with previous attacks of slight jaundice, accompanied by painful enlargement of the liver, is quite sufficient to settle the diagnosis in most cases. Where the diseased organ is already contracted so as to be beyond the reach of palpation, inequalities on its surface afford no help, and, owing to the ascent of the diaphragm, it may be difficult to determine the extent of the hepatic dulness. In such instances, especially where evidence of an alcoholic history is suppressed, the diagnosis cannot be established till the ascitic fluid is removed, when the enlarged spleen affords very valuable aid. The fluid withdrawn in the ascites of atrophic cirrhosis may show by its relatively small amount of albumin, its low s.g., and absence of marked tendency to coagulate, that there is no inflammation of the peritoneal membrane. The absence of cancer-cells and tubercle-bacilli in a centrifugalised specimen of the fluid is also of importance.

Carcinoma of the liver is only likely to be mistaken for a cirrhosis in which the organ is still enlarged, and if the cancer is of the infiltrating variety the differentiation presents difficulties which can only be removed by a careful investigation of the history and by a painstaking search for other evidence of alcoholism, as neuritis, &c. Nodular cancer leads to the formation of irregular protuberances, which may be umbilicated and are of larger dimensions than the hobnails of cirrhosis; they may be felt to increase almost from day to day in rapid cases. When jaundice supervenes it becomes of the dark-green type, the stools are white, the gall-bladder distended, and the spleen is not enlarged. Cancer of the liver causes death generally within a year of its onset, whilst cirrhosis from alcohol may exist for years even when associated with much wasting.

Tubercular peritonitis may produce such enlargement of the rolled-up omentum as simulates an enlarged liver, and

the ascites may prevent this being mapped out. The presence of tubercle elsewhere, and the fever which accompanies it, will usually settle the diagnosis; but when this is impossible, tapping will demonstrate the characteristic infiltration of the omentum and lymphatic glands. It must not be forgotten that abdominal and general tuberculosis is not an infrequent complication of atrophic cirrhosis.

Thrombosis of the portal vein may be recognised by the more rapid onset and progress of the ascites following some inflammatory condition, evidence of the etiology of which may be forthcoming.

Syphilitic hepatitis which has produced gummata will be differentiated by the larger size of its irregular nodular outgrowths, and by the splenic tumour being of large dimensions. The ascites may vary in extent from time to time or disappear, and a history of syphilis and the evidence of the disease in other organs will settle the diagnosis.

Prognosis.—If the diseased condition could be recognised in the early stage, before portal obstruction has become marked, there is every reason to believe that complete recovery would follow upon cessation of spirit-drinking. Alcoholic cirrhosis is practically never seen in reformed drunkards who have given up drinking before liver-symptoms have appeared; the disease is almost always found in patients whose history shows that they have continued to take alcohol up to the time of the supervention of the first signs of portal cirrhosis. This fact goes far to prove that the disease, like that of amyloid degeneration, may in its earlier stages entirely disappear after the removal of the cause.

As regards the prognosis, once the signs of portal obstruction have become obvious, it must always be regarded as most grave the great majority of ascitic cases die within a few months after the fluid has caused distension of the abdomen. Occasionally a patient has been reported as recovering after tapping, but such result is never to be counted upon. In patients seen during the stage of enlargement without ascites the duration may extend to years, as there is a tendency for the disease at this stage to remain stationary, possibly owing to the successful establishment of the collateral circulation. It must be remembered that an advanced degree of alcoholic cirrhosis has been met with in patients who have died from other causes and who have never exhibited any hepatic symptoms. Whether such a result is to be attributed to the collateral circulation, or whether these cases would have ultimately succumbed to the effects of a progressive cicatricial shrinkage of the organ, must at present remain doubtful.

Treatment.—The obvious indication is the removal of the cause—the use of concentrated alcoholic liquors. The digestive troubles, hæmorrhages, &c., must be treated upon

general lines and suitable liquid dietary. Drugs are of little avail; the cirrhotic change is uninfluenced by iodides, arsenic, or other alteratives, but considerable benefit, for a time, follows the use of saline purgatives and stimulating diuretics.

Ascites must be relieved when the abdomen becomes distended. This can only be achieved by the use of the trocar and cannula, as described upon page 82. The operation may require repetition at short intervals, as the fluid rapidly accumulates when the atrophy is marked. The use of dry diet, purging, and diuretics have but slight influence in retarding the rate of accumulation.

Since life may be prolonged for an indefinite period when the collateral circulation is sufficiently perfected by the *vis medicatrix naturæ*, there is a shadow of a hope that recovery may set in even after repeated tappings; but unless the process be assisted by surgical measures no such result can be counted upon.

Talma's operation of epiplopexy or omentopexy—by means of which the omentum is stitched to the curetted abdominal peritoneal surface so as to produce extensive adhesions and a vascular connection between the portal and systemic vessels—promises to become the routine treatment of alcoholic cirrhosis in the future. Nearly 40 per cent. of recoveries may be expected, according to the most recent statistics, when the operation is not delayed till the advent of toxæmic symptoms.

Sinclair White has collected 227 cases in which epiplopexy has been performed for the ascites of hepatic cirrhosis. Of these 37·3 per cent. were cured, 15 per cent. failed, 13 per cent. improved, and 33 per cent. died. He advocates a trial of the method by continuous drainage before resorting to the more serious operation, and he emphasises that absolute abstinence from alcohol is essential after the operation.

HYPERTROPHIC CIRRHOSIS

This is a distinct affection, which should not be confounded with the enlargement of the liver found in the early stages of atrophic cirrhosis. It is often called Hanot's disease, and is sometimes described as *Unilobular* or *Insular* cirrhosis, because the new fibrous tissue-growth is seen to surround the individual liver-lobules, in contrast to the atrophic form, which is frequently called *Multilobular* cirrhosis, because the fibrous tissue encloses or surrounds the remains of many lobules. It has also been described as *Biliary Cirrhosis* on account of the richness of the fibroid tissue in newly formed bile-ducts and the thickening of the walls of the biliary passages, in contrast to the atrophic form, which is sometimes called *Portal Cirrhosis*.

Etiology.—Unlike that of atrophic cirrhosis, it is doubtful if alcohol plays any or but a minor *rôle* in the production of this disease, though some authorities regard alcoholism as a predisposing cause. In the great majority of cases an alcoholic history is absent. It is much rarer than the atrophic form, and is very seldom seen in females, most of the instances being in young men of between twenty and thirty, many being in boys, and some in children. A parasitic origin is suspected, but there is no demonstrable etiological factor present.

Morbid Anatomy.—The liver is greatly enlarged, and may weigh up to 8 lb. ; the enlargement is uniform, and the surface smooth or only marked by small granulations, which are usually of the same size everywhere. The colour is a dark mottled green, and the diseased organ is uniformly increased in firmness and elasticity. Broad strands of fibrous tissue are seen to surround or isolate the lobules ; numerous round cells are visible in the fibroid proliferation in microscopic sections, the strands of which are observed to pass into the lobules dividing the hepatic cells. This fibroid growth does not cause destruction of the liver-cells, which are often seen to be increased in size, and may be multinuclear. The bile-ducts are free, though their walls are greatly thickened, and, as stated above, numerous biliary passages are visible in the newly formed fibrous tissue. The coats of the portal and hepatic blood-vessels are normal, contrasting with the fibrous infiltration around the coats of the portal vein observed in atrophic cirrhosis.

The spleen is usually much enlarged, but this cannot be attributed to stasis ; it is hard, and its capsule shows signs of chronic inflammatory changes.

Symptoms.—The early symptoms are like those seen in atrophic cirrhosis—dyspepsia, nausea, or morning vomiting, with transient attacks of mild jaundice, associated with some fleeting hepatic pain, tenderness, and enlargement. Ultimately the hepatic swelling steadily progresses ; and the next attack of jaundice, instead of passing away, remains permanent. The skin becomes stained of a deep lemon-yellow ; the stools, however, always contain bile, and the urine is also deeply bile-stained. An evening rise of temperature is very frequently present, and marked leucocytosis is a usual feature.

The enlarged organ is felt occupying a considerable proportion of the abdominal space ; its lower margin feels firm and resistant, and may extend much below the umbilicus. The surface is smooth or but indistinctly irregular or granular, and the entire organ is tender and painful. The ribs on the right side often show marked bulging, caused by the pressure of the enlarged liver ; and the spleen, *at an early*

stage, is felt to be much increased in size and hardness, and it is both painful and tender to the touch. The gall-bladder is not distended.

At this advanced stage, with its deep jaundice and absence of ascites, the case contrasts markedly with atrophic cirrhosis, associated as this latter disease always is with marked portal obstruction, and often with but a faint icteroid tinge of the skin and great distension of the superficial veins of the abdomen.

There is marked tendency to hæmorrhages, and toxæmic symptoms supervene as the jaundice deepens, so that the patient may present the clinical picture of malignant jaundice, associated with more or less fever.

The duration of the disease may extend from the commencement of the transient attacks of jaundice, liver-pain, and swelling, to a period of several years, before fatal hæmorrhage, asthenia, or coma supervenes.

Though of essentially chronic progress, the malady may in some cases present stages of apparent improvement, whilst in others it may be steadily progressive. Emaciation always supervenes, though the appetite returns, and in the jaundiced stage the digestion may appear unusually active and stomach-symptoms be entirely absent. Cardiac degeneration ultimately sets in, and towards the close some fluid may be detected in the peritoneal cavity, the result of repeated attacks of peritonitis; but the ascitic distension constantly met with in the atrophic disease is never witnessed. In the case of children, the splenic enlargement is a marked feature, and clubbing of the finger-tips and swelling of the articular ends of the femur and tibia may be observed.

Diagnosis.—From *atrophic cirrhosis* the disease is differentiated by the contrasts in the symptoms already mentioned, chief of which are the presence of severe jaundice, the absence of ascites, of enlarged superficial veins, and other signs of portal obstruction, together with the presence of early swelling of the spleen, and often an absence of any alcoholic history. The age of the patient is also an important point in the diagnosis, as many of the subjects of hypertrophic cirrhosis are children or young adults.

From the enlargement of the liver met with in *mitral disease* the diagnosis is easy, on account of the ascitic fluid present in that affection, together with the signs of the valvular lesion and absence of marked splenic enlargement. From *carcinoma* of the liver hypertrophic cirrhosis is easily differentiated by its chronic course and by the uniform nature of the enlargement.

Swelling of the liver with jaundice caused by *gall-stones* and biliary obstruction is differentiated by its history and by the presence of the distended gall-bladder. It must be

remembered that incomplete obstruction of the common duct, with infective cholangitis, may lead to jaundice, with some bile in the stools and enlargement of the spleen ; but though the jaundice in such cases may remain for a long period, the enlargement of the liver never reaches the dimensions seen in hypertrophic cirrhosis.

Banti's disease is the name given to that form of splenomegaly which is found in combination with hepatic cirrhosis ; the anæmia is marked and of the secondary type seen in splenic anæmia. (See under 'Spleen' and 'Anæmia.')

Prognosis.—The disease is always fatal ; any recoveries reported are probably instances of mistaken diagnosis. The duration may, however, be lengthened out to a period as long as seven years ; though in cases where the enlargement has not been noticed till after the supervention of permanent jaundice the course of the malady may appear as short as in malignant disease of the liver.

Treatment.—Drugs and all known agents are useless, except for the alleviation of symptoms, which must be treated upon the recognised lines indicated in each case. Thus, early vomiting may be relieved by gastric sedatives, and hæmorrhages by the administration of calcium chloride.

CIRRHOSIS OF THE LIVER FROM OBSTRUCTION OF THE BILE-DUCTS

As already stated, hypertrophic cirrhosis is sometimes designated Biliary Cirrhosis, because of the changes observed in the walls of the bile-passages. The form of cirrhotic change which results from the irritation of bile caused by chronic obstruction of the bile-ducts is of little clinical interest. The condition is usually associated with gall-stones, but may follow obstruction of the main ducts by cancerous growths and cicatricial narrowing. The growth of fibroid tissue caused by the irritation of bile as a rule seldom leads to shrinking of the organ, and signs of portal obstruction are rarely present, the symptoms produced by the blocking of the ducts entirely overshadowing those produced by the influence of the bile upon the hepatic tissue. The condition, therefore, is one mainly, if not entirely, of pathological interest, and the microscopical characters are those of ordinary cirrhosis.

The same remarks apply to the fibroid proliferation which is found in the enlarged liver produced by chronic hyperæmia the result of mitral and chronic pulmonary lesions, and which is sometimes designated as *Cardiac Cirrhosis.*

Malarial and *Diabetic Cirrhosis*, save in the fact that it is usually characterised by the deposition of a considerable amount of pigmentation caused by changes in the hæmoglobin of the blood, is of little importance.

Capsular Cirrhosis is a term sometimes applied to chronic perihepatitis, in which disease the proliferation of the fibroid tissue may extend from the capsule into that of the interstitial hepatic tissue, and it will be found described under 'Perihepatitis.'

LIVER, CONGESTION OF

Hyperæmia of the liver is either *active* or *passive*. The amount of blood in the liver varies enormously in health, and is greatly increased during digestion after heavy meals.

ACTIVE CONGESTION

Etiology.—This condition is usually the result of over-eating, especially when the intervals between meals are shortened. The nature of the food consumed plays an important part, and the use of alcohol, condiments, and concentrated or highly seasoned foods tends to induce active congestion by inducing a catarrhal condition of the stomach and intestinal mucous surface. The plethoric state induced by over-feeding, associated as it nearly always is with diminution in the amount of active muscular exercise and lessening of the respiratory movements, still further increases the tendency to the accumulation of blood in the organ.

The influence of a high external temperature is usually quoted as an important etiological factor, since active hepatic congestion is so commonly observed amongst Europeans residing in the tropics ; but the explanation of the coincidence must be sought for in the hygiene of the individual. The amount of food and alcohol which is habitually ingested without inconvenience and danger in temperate climates, if consumed by the same individual on his removal to a tropical region, is almost certain to induce hepatic congestion by causing a catarrhal condition of the digestive tract which seriously interferes with or modifies the digestive processes. Moreover, the influences of the toxins which are manufactured by the parasites causing malaria, dysentery, and other tropical feverish states, are at the same time operative.

Similar toxæmic influences may occasionally be observed in temperate climates in those cases where active congestion of the liver follows typhoid or other infectious fevers, summer diarrhœa, &c.

Rapid reduction of temperature, by causing chilling of the external surface of the body, may induce an active but transient hepatic engorgement ; and a similar condition is believed sometimes to occur upon the cessation of attacks of repeated bleedings from piles, or during menstruation which has been checked or suppressed.

Symptoms.—These are not well defined, and they vary with the cause which has produced the hyperæmia. In their most typical form they are witnessed in the transient prodromata met with prior to the development of alcoholic cirrhosis. Nausea the result of gastric catarrh, flatulence, foul tongue, headache, a sense of oppression in the epigastrium and often pain referred to the right shoulder, and a transient icteroid tinge of the conjunctiva or sallowness of the skin, direct attention to the liver.

The organ will be found somewhat enlarged, and always more or less tender to pressure, and usually some degree of increased firmness may be detected in its consistence. These symptoms and signs are often only present after eating, and may disappear on fasting. The urine is scanty and concentrated, depositing much urates on cooling. If the error in diet has been such as to produce a smart intestinal catarrh, copious diarrhœa follows, with speedy amelioration of the symptoms; more frequently constipation results. Should the cause remain operative, or dietetic indiscretions be continuous, the jaundice increases, the hepatic enlargement becomes more marked, and the tenderness and liver-pain cause considerable inconvenience and suffering.

This diseased condition never leads to a fatal issue unless acute hepatitis supervenes from the operation of some tropical infective organism or chemical poison; hence its pathological anatomy is not demonstrable. Probably a general vascular engorgement of the organ would be all that would be visible.

Diagnosis.—This rests upon the transient nature of the attack following dietetic errors, which manifest themselves by the symptoms of gastric catarrh already detailed. The persistence of signs of hepatic enlargement and jaundice should be interpreted as evidence that some structural alteration of a suppurative or cirrhotic nature has supervened, which puts the case out of the category of active congestion.

Prognosis.—The removal of the cause always leads to the speedy disappearance of the hyperæmia.

Treatment.—The discovery of the etiological factor and its immediate correction constitute the first and, in many instances, the only step necessary for the recovery of the patient. The diet should be restricted, and only diluted foods permitted, as vegetable soups, farinaceous dishes, and fish. Cholagogue purgatives, as calomel, followed by salines, should be daily administered for a short period, and a course of the dilute nitro-hydrochloric acid should be continued for a few weeks.

PASSIVE CONGESTION OF THE LIVER

This is the result of mechanical impediments to the outflow of blood from the hepatic veins.

Etiology. — The common causes are chronic valvular disease, especially mitral insufficiency, which produces in its later stages an overloaded state of the right heart. Any valvular lesion which ultimately leads to relative tricuspid regurgitation acts in the same way. Degenerative changes in the cardiac muscle which produce dilatation of the chambers of the heart cause stasis of the blood in the inferior vena cava, followed by increased pressure in the hepatic veins. Pulmonary chronic lesions, as emphysema, chronic bronchitis, extensive pleuritic adhesions, and interstitial pneumonia, by obstructing the pulmonary circulation, cause overloading of the right ventricle and auricle, and produce a similar result. Aneurismal and other tumours may compress the inferior cava directly, or, by impeding the pulmonary circulation, they may increase the blood-pressure in the vena cava and hepatic veins through the engorgement of the right side of the heart. Tight-lacing may induce a considerable degree of passive congestion of the liver.

Morbid Anatomy. — The liver is enlarged, sometimes to a great extent; it is of a deep-red or mahogany tint, and firm. Its section exhibits the well-known mottled or 'nutmeg' appearance, owing to the engorgement of the hepatic capillaries, which causes a deep staining in the centre of each lobule, contrasting with the fatty and pale liver-cells which constitute the peripheral portion of the lobule. In cases of long standing, some induration and atrophic changes usually supervene, owing to the proliferation of the fibrous tissue of the organ. Such a result has led to the description of a special form of interstitial hepatitis under the name of 'cardiac cirrhosis of the liver.'

Symptoms. — The clinical picture presents the obvious cardiac or pulmonary features which belong to the disease causing the increased blood-pressure in the vena cava and hepatic veins. The liver swells, and forms a palpable tumour which may extend downwards below the level of the umbilicus. Occasionally visible pulsation is apparent in the earlier stages, but as the passive hyperæmia becomes permanently established the organ increases in consistence and pulsation disappears.

There is a sense of dragging and weight experienced in the right hypochondrium; sometimes pain is felt in the right shoulder, and usually the enlarged organ is tender to pressure. Owing to the stasis influencing the portal system, gastric and intestinal congestion produces a catarrhal condition, in which symptoms of nausea, indigestion, and hæmatemesis may appear, or hæmorrhoids develop. Jaundice is commonly present; usually it is not of a deep tint, but occasionally it may stain the dusky skin and the conjunctiva a deep lemon or olive colour. The motions are clay-coloured and the urine bile-stained.

Ascites, part of a general dropsy, may appear at a comparatively early stage, when it will be trifling in degree and may become absorbed ; but when the portal blocking is severe in long-standing cases, in which the liver has become indurated, it may be extensive and permanent, unless tapping be resorted to. Enlargement of the spleen, so constantly observed in atrophic cirrhosis of the liver, is seldom witnessed, though a considerable degree of induration of the organ is always found after death. The duration of the condition depends upon the permanence or the transient nature of the primary cause ; and since this is so commonly a mitral lesion, the enlargement of the liver, when once established, remains till the patient's death.

Diagnosis.—The chief point in the recognition of passive hepatic congestion is the evidence of the presence of the primary lesion. The liver-enlargement is distinguishable from all other uniform, smooth, painful enlargements by the variations which from time to time occur in its bulk, the swollen organ appreciably shrinking or increasing in volume as the heart's force fails, or improves under rest and treatment. The liver in children yields very rapidly to backward pressure in the vena cava inferior, and as quickly returns to its former bulk when the cause is removed.

Atrophic cirrhosis, in its early stage of enlargement, may be mistaken for passive hyperæmia ; but the alcoholic history, enlarged veins, marked splenic enlargement, and absence of valvular lesions are characteristic, save in those cases where both conditions co-exist.

Prognosis.—The outlook, once the organ has become markedly enlarged, is grave, since nearly all the factors which induce passive hyperæmia of the liver are irremovable, and eventually end in death. A very considerable degree of improvement sometimes follows treatment in young subjects where compensation can be established by rest, cardiac tonics, and other appropriate measures.

Treatment.—In the great majority of cases the remedies mentioned as suitable for the treatment of failing compensation are indicated, and need not be again enumerated. Where the etiological factor is situated in the pulmonary circuit, the same measures are applicable, since the best results may be hoped for by agents which increase the force of the cardiac muscle. Where the engorgement of the right side of the heart is threatening the general circulation, and the left ventricle is unable to propel the blood through the systemic vessels owing to the abnormal pressure in the veins, a smart blood-letting may avert a fatal issue and afford great relief. Calomel, in full doses, followed by saline purgatives, seldom fails to relieve portal stasis for a time. Diuretics are uncertain, and only those which, like the members of the digitalis group,

operate by increasing the blood-pressure in the renal glomeruli need be relied upon. As a rule, no benefits are to be expected by the withdrawal of liquids from the dietary.

When the ascites becomes extensive, repeated tapping may be resorted to; thus in one case of mitral disease the writer was enabled to prolong a young patient's life for several years by tapping the ascites more than twenty times.

LIVER, ENLARGEMENTS OF

These are described under their respective headings, but an enumeration of the group of diseases causing marked enlargement of the liver is convenient for diagnostic purposes. The list includes the following: (1) Active congestion; (2) Passive congestion; (3) Alcoholic cirrhosis in its early stages; (4) Hypertrophic cirrhosis; (5) Inflammation of the bile-ducts; (6) Obstruction of the ductus communis; (7) Abscesses, both tropical and pyæmic; (8) Carcinoma; (9) Amyloid disease; (10) Fatty infiltration and fatty degeneration; (11) Hydatid disease; (12) Sarcoma, simple cysts, tubercle, and gummata.

Bright divided the group into two divisions, according to the character of the surface-enlargement, whether this was *smooth* or *irregular*. Murchison's classification is of greater clinical importance. He divided the hepatic enlargements into two groups: (1) The *painless*, as amyloid, fatty, hydatid, and the rare and doubtful variety designated 'simple hypertrophy'; the members of this group, in addition to their painless character, are also essentially chronic in their course, and ascites and jaundice are absent. (2) The *painful* enlargements include the remaining members of the list; these run a more rapid course, and are usually associated with ascites and jaundice.

Murchison included in his list of *contractions* of the liver: (1) Simple atrophy, as met with in senility, inanition, and external pressure; (2) Acute yellow atrophy; (3) Chronic atrophy caused by alcohol, prolonged passive congestion, perihepatitis, and the so-called red atrophy of Rokitansky.

LIVER, FATTY DISEASE OF

In the normal liver, fat is always found in the hepatic cells in considerable amount; one-twentieth part of the organ's weight is caused by these deposits, which are stored up in its cells after eating to be given out slowly according to the needs of the body.

Clinically, two varieties of fatty liver are to be recognised. In the one which is termed '*Fatty Infiltration of the Liver*'

the condition is an exaggeration of the normal state, in which fatty globules are found in abundance in the hepatic cells a couple of hours after a meal, the cells themselves being normal and their protoplasm intact.

Fatty Degeneration of the Liver is the name reserved for that condition in which, with the presence of fat in the cells, there are degenerative changes in the cell-protoplasm, which ultimately becomes replaced by fat after the degeneration of the cell-nuclei.

Etiology.—*Fatty infiltration* may be the result of over-feeding, especially with carbohydrates and fat. With this cause, as seen in general obesity, there is usually another associated—viz. deficient open-air exercise. Both these causes are most actively operative about the middle period of life, so that the liver-cells become permanently loaded with enormous quantities of fat.

Anæmia, chlorosis, and phthisis lead to deposition of fat in the liver-cells, it is supposed, owing to retardation of the oxidation-process in the tissues ; and in chronic alcoholism fatty infiltration is constantly observed. In this latter condition the deficient metabolism of fats and carbohydrates is probably the result of the oxygen being used up by the alcohol in its own combustion, as maintained by Hoppe-Seyler. It cannot, however, be disputed that both in fatty infiltration and fatty degeneration of the liver the deposition of fat in the organ, in many instances, can be explained by the transference of the subcutaneous adipose material to the liver-cells. A similar explanation is forthcoming in those cases where fatty degeneration of the muscles and other organs in acute fevers, phthisis, cancer, diphtheria, marasmus, &c., leads to the storing up in the liver of the fat which exists in excess in the blood.

In passive hepatic congestion, the result of valvular lesions, fatty infiltration and sometimes fatty degeneration, of the peripheral portions of the lobules are visible.

True *fatty degeneration* of the liver-parenchyma is seen in its most typical form in phosphorus-poisoning, where the fatty infiltration rapidly passes into degeneration, destroying the protoplasm of the cells, which is more or less completely replaced by free fat. Identical results, though more slowly produced, are witnessed as the effects of poisoning by arsenic, antimony, and other substances.

Acute yellow atrophy of the liver shows essentially the same degeneration of the hepatic cells and their replacement by fat.

Pathological Anatomy.—The organ in *fatty infiltration* is greatly enlarged, and may weigh 7 or 8 lb. Its surface is smooth and its margins rounded ; the colour is paler than the normal, being yellow, and apparently bloodless. The

consistence is somewhat increased, the organ feels doughy, and when removed from the body may float upon water; the enlargement is always uniform. On section it cuts readily, greasing the knife and fingers. The microscopic characters are distinctive; the cells and their nuclei are normal, but they may be invisible owing to the great infiltration of the lobules by fat-globules everywhere.

In *fatty degeneration* the pathological changes are those already described as characteristic of acute yellow atrophy. Jaundice being usually present, the organ is deeply bile-stained —a condition never seen in fatty infiltration, unless some cirrhotic change is associated with it. In very acute poisoning by phosphorus the liver will be somewhat enlarged, and if death has occurred early the changes may be entirely the result of infiltration; but in cases of some standing the destruction of the cells and their nuclei, and their replacement by fatty globules, reveal a true degeneration, with shrinking in the volume of the organ.

Symptoms. — In *fatty infiltration* the liver-parenchyma being intact, there are no definite hepatic symptoms, except those caused by the mechanical inconvenience of an enlarged organ in the right hypochondrium. The clinical features are those of the condition to which the fatty infiltration is secondary; as this is very often a state of general obesity the enlargement cannot be easily made out.

In phthisis and chronic alcoholism the thinness of the abdominal parietes permits of the enlargement being recognised without difficulty. The uniformity of the increase in all the diameters of the liver may be mapped out by percussion; and palpation reveals a smooth outline, blunted or rounded margin, and a soft doughy feel to the fingers.

Though the fatty liver seems increased in consistence when examined after death, its soft and yielding feel to the touch easily distinguishes it from amyloid disease when palpated during life through a thin-walled abdomen; both conditions are painless.

There are no symptoms or signs of portal obstruction. Jaundice is never present, but the stools may be light-coloured from deficient production of bile. The duration and course of the primary disease producing the fatty infiltration is apparently neither hastened nor retarded by the involvement of the liver.

When fatty infiltration occurs in chronic alcoholism the signs and symptoms may become mixed with those of cirrhosis, though a fatty liver in a drunkard is occasionally met with which exhibits no interstitial fibroid proliferation. Such an organ will reveal its presence by the smoothness of its surface, the softness of its feel to the fingers, and by the absence of tenderness, jaundice, and portal stasis.

When cirrhosis supervenes the smooth outline gives way to an irregular lower margin, there is increased resistance and tenderness, and an icteroid tinge in the conjunctiva follows symptoms of portal stasis, with splenic enlargement and distension of the superficial veins.

The symptoms of acute fatty degeneration of the liver are those already detailed under 'Acute Yellow Atrophy,' and need not be here repeated.

Diagnosis.—The various points in the differentiation of fatty liver from *amyloid degeneration* and from *cirrhosis* have been already sufficiently touched upon in the preceding page. The degeneration following *phosphorus-poisoning* is differentiated from acute yellow atrophy mainly by the history of the ingestion of the poison, which produces an immediate gastritis, followed after a pause of two or three days by jaundice, without fever.

Prognosis.—This is the forecast of the primary disease, fatty infiltration of the liver practically causing no intensification of the symptoms. When fatty degeneration of the heart-muscle is present, either as a cause or result of the liver-affection, the prognosis becomes grave. In fatty degeneration of the liver the outlook obviously possesses the gravity of the fatal toxæmia which has caused it.

Treatment.—This resolves itself into the management of the general obesity, alcoholism, anæmia, or cachexia present, by a careful regulation of the dietary, a readjustment of the hours of rest, exercise, &c. In poisoning by phosphorus and other substances antidotes are of little avail, as the liver-condition is already established before symptoms of it appear; but it is well to remember that particles of phosphorus may be found in the intestines several days after the poison has been swallowed, so that free purgation, combined with turpentine administration, should be resorted to when the case comes under notice at an early stage. In phthisical patients the question of over-feeding and the administration of animal fats and alcohol should receive careful consideration, as it may be doubtful to what extent these agents tend in some cases to prevent the transference of the fat of the subcutaneous tissue to the liver-cells.

LIVER, FUNCTIONAL AFFECTIONS OF

That the various functions of the liver may be diminished, increased, or perverted in the course of organic disease of the organ has been incidentally referred to in the description of the symptoms met with in its different structural affections.

The disturbance of the glycogenic function of the liver, and its supposed relationship to the causation of glycosuria,

have been discussed under 'Diabetes.' Murchison attached such importance to the disturbances of the functions of the liver that he described no less than nine different conditions, or groups of symptoms, arising from this cause. By modern authorities most of these symptoms are now regarded as having their origin in other organs, especially in the stomach and intestines, where, owing to catarrhal or other conditions, auto-infection is produced by the absorption of toxins which depress the nerve-centres and cause the disturbances formerly held to be characteristic of disturbed hepatic function. This change of view is accountable for the fact that in many modern text-books on medicine no reference whatever is made to functional affections of the liver.

The theory that *lithæmia* is to be regarded as the result of a depression of the metabolic function of the liver has still something to be said in its favour, and its importance in association with the production of gout does not require accentuation. Owing to some congenital or acquired defect in the efficiency of the liver-cells to convert nitrogenous material into urea, the intermediate products, whether in the form of uric acid or other toxic substances, may accumulate in the blood to the extent of causing considerable injury to other organs. The more modern view of the production of uric acid from the nuclein of the leucocytes only partially transfers the explanation of the pathology of the uric-acid diathesis to leucocytosis.

Uneasiness and oppression felt in the hypochondriac region, sallowness, dyspepsia, flatulence, nausea, and furred tongue, with bitter taste in the mouth, headache, drowsiness, or irritability of temper, depression of spirits, and cardiac irregularity, accompanied usually with copious deposits of urates in the cooled urine, are popularly regarded as symptoms of torpidity of the liver or biliousness.

As these symptoms so commonly follow excesses in eating and drinking, the question of whether they are to be regarded as evidence of a toxæmia the result of hepatic insufficiency, or as caused by catarrh of the stomach which extends to the bile-ducts, must at present remain doubtful. One thing, however, seems highly probable, that a continual repetition of the error in diet leads to the condition described under 'Acute Congestion of the Liver.'

The fact that in many structural diseases of the liver, associated with considerable wasting or destruction of its parenchymatous tissue, the above-mentioned symptoms do not appear is of itself strong presumptive evidence that the origin cannot be with certainty attributed to mere hepatic insufficiency.

Disturbances of the biliary function of the liver or of its bile-secreting canals were formerly likewise credited with

the production of various symptoms; but the fact that bile plays but an insignificant part in the digestive process robs this theory of its ancient importance. That symptoms like those previously mentioned are sometimes associated with jaundice is easily explained on the assumption that, along with the absorbed bile, toxins and other products, the result of arrested metamorphosis, pass into the circulation at the same time.

Treatment.—Whatever difficulty may be encountered in attributing the symptoms usually known as biliousness to those of a primary functional disturbance of the liver, there cannot be any difficulty about their treatment. This must be directed to the etiological factor; and since this in the great majority of cases is obviously the result of errors in dietary, the agents mentioned as suitable for the prevention and relief of acute congestion of the liver are clearly indicated.

LIVER, HYDATID DISEASE OF. (See p. 652.)

LIVER, INFLAMMATION OF

Acute hepatitis affecting a limited area of the hepatic substance is seen in its typical form in the conditions which end in suppuration, as described under 'Liver Abscess' (p. 818). The inflammatory process, whether manifesting itself as a tropical hepatitis, portal pyæmia, part of a general pyæmic condition, or an infective cholangitis, is almost invariably the direct result of the introduction of micro-organisms, which lead to the same results as are observed in suppuration in other regions of the body.

Acute diffuse parenchymatous hepatitis is a frequent secondary result of the chemical poisons manufactured by the organisms which produce typhoid fever, septicæmia, erysipelas, diphtheria, malaria, dysentery, and several other affections. The acute congestion of the liver so frequently met with in the tropics may pass into this condition.

The pathological changes consist in a cloudy swelling of the hepatic cells, owing to the presence of fine granules: the cells lose their definite outline, and their protoplasm and nuclei cease to react to the ordinary staining re-agents.

The symptoms are mainly those of the primary disease, and the condition is usually unsuspected during life, except in those cases of tropical hyperæmia where the swelling and pain of the organ direct attention to the right hypochondrium; but even then a differential diagnosis between active congestion and a parenchymatous hepatitis is impossible.

An *acute interstitial hepatitis*, in which the parenchymatous inflammation is associated with proliferation of the fibroid tissue of the liver, is recognised by some authorities. It is, however, impossible to differentiate such cases from acute yellow atrophy and from ordinary cirrhosis which runs an unusually rapid course. Acute yellow atrophy itself, already described under its own title, must be regarded as a form of acute hepatitis, probably of the diffuse parenchymatous type, ending in destruction of the liver-cells, and usually associated with rapid proliferation of the interstitial connective tissue.

Chronic hepatitis, as a sequel of the acute diffuse parenchymatous type, or commencing as a chronic affection, cannot be regarded as of clinical interest. If such a condition exists, it can only be of purely pathological interest. For all practical purposes the essential feature in all forms of chronic inflammation of the liver, so-called, is the obviously resulting change produced by the proliferation of the fibroid tissue of the organ. Hence chronic inflammation of the liver is a synonym for chronic interstitial hepatitis or cirrhosis, whether of the atrophic or hypertrophic variety. These conditions are described under 'Cirrhosis,' and the chronic interstitial affections which result from syphilis, perihepatitis, &c., will be found under their own headings.

PERIHEPATITIS

Acute inflammation of the serous coating of the liver is a common event in all inflammatory states of the general peritoneum, and possesses little clinical interest.

When the process is of chronic nature it will be found that the areolar or fibrous capsule which intervenes between the peritoneal covering and the glandular substance is involved. This fibrous coat is intimately connected with the fine areolar tissue which divides the hepatic lobules; at the transverse fissure of the liver it affords a strong investing sheath for the vessels and duct and their ramifications, and forms the capsule of Glisson.

Etiology and Pathology.—Chronic inflammation of this areolar coat and the investing peritoneum is usually secondary to some hepatic disease, as cirrhosis, syphilis of the organ, abscesses, parasitic or other growths; but occasionally it may be the result of an adhesive inflammatory process starting in the gall-bladder or in some organ in the immediate vicinity of the liver, as in the right pleura or pericardium, or in the neighbourhood of a gastric ulcer which has become glued to the hepatic capsule.

It is usually more or less obviously present in a localised form in the malformations produced by the pressure of tight-lacing. In a diffuse type of the disease in which the entire

capsule of the liver becomes greatly thickened by the proliferation of its fibroid tissue, so as to surround the organ with a dense envelope which may measure more than a third of an inch in section, there may be witnessed much atrophy of the parenchymatous tissue. This form of capsulitis, described by Hübler, Pick, and others, is found associated with the presence of gall-stones or adhesive pericarditis; and since similar changes are very frequently present in the spleen and kidney, Hale White maintains that it is secondary to interstitial nephritis. Often the interstitial fibroid tissue of the liver escapes change, but sometimes a general cirrhosis of the organ supervenes.

Symptoms.—In the acute cases involving the peritoneal coating of the liver the symptoms are those of the primary inflammation of the peritoneum, which generally overshadow the capsulitis.

In the chronic diffuse form the pressure of the thickened capsule, which progressively tends to shrink as its fibroid tissue contracts, ultimately interferes with the portal circulation, so that all the clinical features of atrophic cirrhosis become developed. Ascites appears, generally unassociated with jaundice, and after tapping the fluid reaccumulates rapidly, requiring repeated withdrawals, which may be indicated at short intervals spread over many years. In this respect chronic perihepatitis is in marked contrast with alcoholic or atrophic cirrhosis, which, though also a chronic disease, generally ends in a fatal issue within a few months after the first tapping has been rendered necessary.

The presence of changes in the urine indicative of interstitial nephritis is regarded by White as of special diagnostic value in the differentiation. The presence of adherent pericardium, fibrinous peritonitis, or extensive pleural thickening, and perisplenitis, is an aid to diagnosis.

The liver, being much reduced in size, is usually beyond the reach of palpation; but when felt, as it may be immediately after a tapping, its comparatively smooth outline and uniformity of surface are in marked contrast to alcoholic cirrhosis.

Prognosis is that of the primary disease; and, as already stated, though the condition is a progressive one, life may be prolonged for many years by repeated tappings. Sometimes the disease, however, makes a halt between the tappings, possibly owing to some partial success of the natural attempt at the establishment of the collateral circulation.

Treatment.—The measures enumerated under 'Cirrhosis' are indicated, symptoms being relieved as they arise by suitable remedies, and tapping being frequently indicated. Probably omentopexy might be followed by permanent improvement in selected cases.

LIVER, MALFORMATIONS OF

Gummata and tumours of a malignant and parasitic nature cause extensive alterations in the form of the liver, which are described in the sections dealing with the primary affections. Congenital malformations are, as a rule, only of museum importance and interest, not being capable of demonstration during life.

Tight-lacing produces an acquired deformity of the liver which is sometimes described as 'corset liver.' The shape of the deformed organ varies considerably, owing to the acuteness of the constricting band in the clothing which causes the pressure, and also owing to the level at which the constriction operates. Where the pressure is evenly diffused by tight-fitting corsets the liver becomes much elongated by the narrowed and deformed thorax, the right lobe being prolonged into a tongue-shaped process, which may extend below the level of the umbilicus, and this portion is marked off from the main part of the organ by a transverse groove.

Where a tight band in the clothing is used to produce a low waist or a high waist, the pressure causes atrophy, which is seen in the transverse sulcus passing across the anterior surface of the organ, and the deformity will be seen to vary with the level of the applied pressure. In extreme cases the prolonged portion may be almost separated from the bulk of the liver by a membranous attachment, which contains no liver-substance, but only vessels and bile-ducts.

Symptoms. — As a rule these are absent, but a physical examination will reveal the malformation. The consistency of the organ is increased, owing to the resulting perihepatitis, and this, with the hyperæmia present, is marked in the portion below the level of the transverse groove.

In the deformity caused by diffuse pressure the elongated liver is forced below the costal arch on the right side, and the tongue-like projecting portion may incline backwards into the flank and simulate a floating kidney. By careful palpation it may, however, be proved to be continuous with the mass of the liver by tracing the left border or margin of the organ from left to right; and it moves freely with respiration. Where the portion of liver has become almost detached from tight waist-band pressure, careful palpation will also usually prove its continuity with the margin of the left lobe; but it is well to remember that the intestines may lie in front of it and give a clear note on percussion.

The malformation may interfere with the patency of the ducts and the flow of bile, so that gall-stones may form, giving rise to pain and other symptoms; and the lacing

may, at the same time, still further complicate matters by causing ptosis of the right kidney and stomach.

Displacement of the liver occurs in every condition in which the diaphragm is either elevated or depressed; in deformities of the thorax and spine the organ may be considerably dislocated.

Movable liver, from the minor degree of hepatoptosis commonly seen in the enteroptosis occurring after repeated pregnancies, to the exaggerated extent recognised as *floating* liver, is also met with. The last-named condition is very rare, but occasionally the movable organ has been found occupying the greater portion of the right side of the abdomen, its lower margin extending to the symphysis pubis. (See under 'Enteroptosis.')

The tongue-like lobes described and named after Riedel resemble the deformity caused by tight-lacing; they are generally, however, the result of distension of the gall-bladder caused by calculi, and they are often so freely movable that they are usually styled 'floating lobes.'

LIVER, SYPHILITIC DISEASE OF

The frequency with which syphilitic hepatitis is found in the bodies of infants who have suffered from *congenital* syphilis renders it very probable that slight syphilitic changes are present in a large percentage of the livers of infants who are labouring under the hereditary form of the disease, though no hepatic signs or symptoms may be present.

The liver of *congenital syphilis* shows the disease in two well-marked types, differing little from the varieties observed in the acquired form of the disease. The most frequent type is that in which the organ is the seat of a diffuse interstitial hepatitis. The fibroid tissue between the lobules proliferates, and the new-growth often penetrates the lobule and extends between the columns of the hepatic cells. The organ is uniformly enlarged, and becomes greatly increased in consistency, so that it cuts with difficulty, being sometimes as dense and tough as sole-leather. With the increase in the fibrous material there is at first abundance of small round cells, which infiltrate the vascular walls.

The hepatic enlargement may be present at birth, or only manifest its presence during the first year of extra-uterine life. The clinical picture is that of a hypertrophic cirrhosis, in which the indurated, smooth, and uniformly enlarged organ may be felt extending below the level of the umbilicus. Ascites and splenic enlargement, with induration, supervene; jaundice is comparatively rare, there is very considerable emaciation, and death often occurs from marasmus owing

to intestinal troubles. When the patient survives, the shrinking of the interstitial fibrosis may lead to considerable wasting of the hepatic tissue, and the clinical picture closely simulates or becomes identical with that seen in atrophic cirrhosis.

The other form of congenital hepatic syphilis is that in which the disease manifests itself in a circumscribed hepatitis, associated with the formation of gummata resembling those found in the acquired disease. Usually, however, the new-growths in the congenital form are more numerous, and may stud the liver over with small or miliary tubercular masses.

When the disease appears late, in the so-called rare 'delayed' or 'retarded' variety, which may not manifest itself for seven or ten years after birth, there is usually much sclerotic change in the capsule, and nodular masses of indurated fibroid growth and old gummata may cause considerable deformity.

Acquired Syphilis.—The secondary stage of the disease is possibly often associated with some diffuse hepatitis ; but, if so, the symptoms are so slight as to escape notice, and resolution constantly takes place. The typical syphilitic liver is a manifestation occurring during the *tertiary* stage of the disease, about the same period in which gummatous growths are observed in the periosteum and other tissues. As in the congenital affection, it may only manifest itself as a diffuse interstitial hepatitis ; but this is rare, the formation of variously sized circumscribed gummatous tumours being the rule.

These may reach enormous dimensions; but usually they are met with as nodular masses from about the size of a cherry to that of a small hen's egg. In colour they are greyish-red or of a dirty-white hue, and their centres are yellow. The outline is irregular, and on section the structure presents a radiating appearance, caused by the extensions of the fibrous outer portion of the gumma, which becomes continuous with the interstitial tissue of the liver. In consistence, the cheesy-yellow central zone is often hard and tough, and may contain calcareous deposit which imparts grittiness on section ; but in other cases the centre may be found in the act of breaking down and softening to a pulpy consistence.

The gummatous growths may be few or numerous, and, though most frequently found near the surface of the liver, they may be met with at any depth in its interior. From the round or spindle-celled growth, which first constitutes the gummatous mass, the fibrous stroma develops in varying proportion according to its age. Ultimately it becomes converted into scar-tissue, which, by its cicatricial shortening, leads to stellate puckering and depressions, causing great

malformations, in which rounded or globular portions of the hepatic tissue may be almost detached from the main mass of the organ. Sometimes the liver-substance is divided deeply into lobulated pieces, in the centres of some of which occasionally dried caseous gummata may be found in various stages of change.

The capsule always suffers more or less, and a certain amount of perihepatitis is invariably present. Even in the rare form of diffuse interstitial hepatitis or syphilitic cirrhosis there are generally some gummata to be found, and not infrequently amyloid degeneration supervenes.

Symptoms. — There may be no suspicion of liver-disease until the cirrhotic changes eventuate in portal obstruction, causing ascites, faint jaundice, and splenic enlargement. The patient's condition will then in most respects correspond to that seen in alcoholic cirrhosis.

Sometimes a large tumour may be felt projecting from the anterior surface or lower margin of the organ, which usually becomes firmly attached to the diaphragm or neighbouring viscera. Such bosses resemble malignant disease ; they are usually hard, irregular in outline, tender or painful, and may cause bulging of the epigastrium. Dyspepsia, diarrhœa, albuminuria, or other symptoms of amyloid degeneration, may first direct attention to the state of the liver.

Diagnosis.—*Cancer* of the liver is differentiated with difficulty in patients seen for the first time ; but the rapid growth of old nodules and the speedy appearance of new ones contrast markedly with the slow development of syphilitic gummata. The depression, which may be palpable upon the summit of a carcinomatous nodule, must not be mistaken as evidence of the softening of a syphilitic growth. There is usually a clear history of syphilis, or else evidence of other tertiary growths in the bones, and the lesion betrays its specific nature by the readiness with which it resolves or vanishes under large doses of the iodides ; the pallor and wasting are seldom so extreme as in cancer, which, moreover, runs its fatal course inside one year.

Alcoholic cirrhosis never presents the large irregular bosses seen in the syphilitic variety ; the disease is rapid after the first advent of ascites, and the splenic enlargement, though marked, is much less obvious than in syphilis ; there may be also other evidences of chronic alcoholism, as neuritis, present. Unfortunately, the frequent association of syphilis and the drink-habit renders a combination of both conditions a likely event, and when amyloid degeneration supervenes a very careful investigation of the patient's past life and habits is necessary to disentangle the symptoms of the triple combination.

Treatment.—Like the question of prognosis, the treatment resolves itself entirely into that of tertiary syphilis. Haubold and Anchütz have recently shown that gummata may be removed surgically with good results; but the percentage of cases requiring such radical surgery must be very small.

LIVER, TUMOURS OF.

(See under LIVER, CANCER OF, p. 833; HYDATID DISEASE, GALL-BLADDER DISEASES, &c.)

LIVER-VESSELS, DISEASES OF

Disease of the portal vein, causing pylephlebitis, either adhesive or suppurative, is described under 'Thrombosis' and 'Liver Abscess.'

LOCOMOTOR ATAXIA. (See under ATAXIA.)

LUMBAGO

This is the name given to a painful affection involving the lumbo-spinal and lumbar muscles and their fibrous sheaths, and usually only evidenced upon attempted movement of the spine.

Etiology.—The attack can commonly be traced to exposure to cold and damp, often associated with muscular fatigue. Men are much more frequently affected than women, and a gouty or rheumatic diathesis is suspected to be present in most cases. The condition is one usually seen after middle-life. Romer has recently pointed out the frequency with which trauma may be observed as the only etiological factor in cases running a chronic course.

Pathology.—The affection has been generally regarded as of the nature of muscular rheumatism, but some physicians maintain that it is a simple neuralgia, notwithstanding the absence of all spontaneous pain in most cases. Gowers has, however, promulgated the theory that lumbago, stiff neck, and pleurodynia, and other forms of so-called myalgia or muscular rheumatism, are due to an inflammation of the interstitial fibrous tissue with which the different nerve-endings (muscle-spindles) are surrounded. Hence he regards these conditions as the result of a *fibrositis* of transient duration, which, however, may become chronic when only tendinous tissue is involved. The invariable absence of a fatal result has prevented any real knowledge being obtained of the actual pathological conditions.

Symptoms.—The onset is sudden, being nearly always excited by some voluntary muscular act necessitating flexion or extension of the spinal column. The patient is seized with violent pain, which compels him to keep the affected muscles in a state of absolute rest, so that if he has been in a stooping or kneeling posture he may find himself quite incapable of straightening his back. The muscles may be tender to pressure, but true spontaneous pain is absent. There is, however, generally a feeling of much discomfort, even when the patient is resting in bed, which compels him to move his body in seeking for an easier posture, when the characteristic pain will be experienced. There is usually no fever or other constitutional disturbance present. The duration of the disease is usually a matter of days, but the affection is liable to return frequently, after which it may become chronic, and last for many months or even years.

Diagnosis.—The violent pain which ushers in *influenza*, *small-pox*, and other toxæmic states may be mistaken for lumbago; but, though such pain is always aggravated by voluntary movements, its spontaneous character is unmistakable, and the fever always evident will also prevent mistakes in diagnosis. *Spinal caries* may closely simulate lumbago of a chronic type; but the spinal symptoms, always to be found when looked for, will demonstrate the true nature of the case. The meningeal pains sometimes present in the early stages of caries are never confined to the lumbar muscles, but follow the course of the spinal nerves.

Disease of the *sacro-iliac joint* can be eliminated by a careful search for its signs and localised symptoms.

Treatment.—The various agents useful for the relief of lumbago will be found detailed upon pages 526-529 of the fourth edition of the author's 'Dictionary of Treatment.' The chief indication for treatment points to the administration of agents which hasten the elimination of waste products by the skin, kidney, and bowel. Diuretics and diaphoretics, like sweet spirit of nitre, gin, salicylates, &c., may be freely used in conjunction with hot baths, warm packs, and fomentations. Iodides in large and, better still, free iodine in small doses are often very efficacious.

Pain should be relieved by deep hypodermics of morphia or cocaine, or by local applications of belladonna, aconite, and chloroform.

In the chronic cases counter-irritation by the cautery and blisters should be tried, and electricity with massage is useful.

Perhaps the best routine treatment in all cases is acupuncture, a stout sterilised needle being deeply driven into the affected muscles at several points. Sir J. A. Grant inserts fine sewing-needles, by means of which he maintains that

striking results are immediately obtained by the withdrawal of charges of pent-up electricity in the involved muscles. In traumatic lumbago, Romer advocates the stretching of the contracted muscles and rupture of the adhesions by forcible movements, executed under chloroform.

LUNG, ABSCESS OF

The suppurating cavities in the pulmonary tissue which are present in phthisis, the sphacelating areæ of pulmonary gangrene, and the pus-containing dilatations which constitute bronchiectasis, are not to be included in the term 'pulmonary abscess.'

Etiology.—A true abscess of the lung may be secondary to acute pneumonia of the croupous type. It is much more common in the form of inflammation of the lung which is produced by the aspiration or deglutition of septic secretions from the upper air-tract after throat-operations, and in cancerous ulcerations, especially when the larynx is paralysed. The writer has seen it follow in submersion cases where the patient had fallen into water contaminated with sewage-matter. It is sometimes the result of the accidental aspiration of foreign bodies, which find their way into a bronchus. Pyæmia is frequently associated with showers of septic emboli, which produce multiple abscesses in the pulmonary tissue.

Wounds of the thorax which perforate the lung may cause the condition. Suppurative processes starting in the pleura, lymphatic glands, bodies of the vertebra, or in liver-hydatids, hepatic abscess, and ulcer of the stomach may extend and perforate the pulmonary tissue, causing a true pulmonary abscess.

Various microbes have been found in the expectorated pus, as the diplococcus of pneumonia, staphylococci, the influenzal bacillus, and many other organisms.

Symptoms.—The multiple abscesses of pyæmia generally remain unrecognised till after death, and usually little suspicion of the abscess-cavity is aroused in any case till the pus is expectorated.

The fever, physical signs, and constitutional disturbance caused by the primary disorder as a rule overshadow the local lesion. The characters of the sputum are of primary importance, and amongst the fragments of broken-down lung-substance the presence of *yellow elastic fibres* settles the diagnosis. In a recent case under the writer's observation a typical croupous pneumonia ended in crisis on the seventh day. A few days afterwards some brownish-black expectoration was brought up. This was followed by a rise of temperature and the expectoration of the contents of an abscess-

cavity, apparently as large as a hen's egg, situated near to the surface in the base of the affected lung, which, after periodical emptyings by coughing, exhibited the typical signs of cavernous breathing. After a few weeks the pleura became involved and a localised empyema resulted, demanding incision and drainage, which were followed by recovery. Probably such cases, coming under observation at a late stage, are mistaken for primary empyemas.

The base of the lung is the usual site of such abscesses, and in pneumonia, where the normal crisis is followed by fever of the septic type, it is well to remember the possibility of a pulmonary abscess, and to have the expectoration examined for elastic tissue should purulent sputum appear, though such an event occurs in but a very trifling percentage of cases.

Prognosis.—It is needless to say that in the metastatic or pyæmic cases death generally results ; but a hopeful prognosis may be given in pneumonic cases suitable for operation.

Treatment.—Inhalations of tarry or other antiseptic solutions or vapours, as practised in the treatment of bronchiectasis and gangrene of the lung, are indicated ; volatile antiseptics should be administered by the mouth at the same time. Surgical measures afford the best prospect of complete cure, and the results are promising in those cases lying under the pulmonary pleura which can be easily reached by a free incision after rib-excision. Aspiration or puncture should not be attempted. Measures directed to the establishment of adhesions between the pulmonary and pleural surfaces are indicated. Cavazzani's recent list of thirteen operations with but three deaths, and the results of Tuffier's 107 cases, prove that the best results are to be expected from *early* operation.

LUNG, ACTINOMYCOSIS OF

The general results of the infective inflammation produced by the ray-fungus or streptothrix, and the description of the parasite, have been dealt with under ' Actinomycosis.'

When the fungus finds its way into the lungs, usually the entrance is effected through the bronchi ; but occasionally these organs may become infected by the extension of necrotic changes arising from actinomycosis of the ribs, sternum, lymphatic glands, or vertebral bodies.

Symptoms.—These may be classed under three types. In the *bronchial* type the symptoms are those of an acute or chronic primary bronchitis in which the ordinary signs of bronchial inflammation are present, the diagnosis only being possible by the demonstration of the ray-shaped fungus in the expectoration, which is often putrid or foul-smelling.

The *pneumonic* form of the disease exhibits the symptoms and signs of a broncho-pneumonia in which there is much increase in the interstitial fibroid tissue of the lung, which is generally studded with abscesses. The clinical picture in this form of the disease closely resembles that of chronic pulmonary tuberculosis, with hectic fever, sweating, emaciation, and profuse expectoration. The freedom of the apices of the lung, the absence of tubercle bacilli, and the presence of the fungus in the sputum are the chief differentiating points in the elimination of phthisis. Pleurisy or empyema may supervene ; the latter is liable to become localised, and to point as an ordinary abscess between the ribs. The pus from such abscesses exhibits the parasite in considerable numbers.

In the third, or *miliary*, type, which is the rarest form of pulmonary actinomycosis, the lung-substance is studded throughout with miliary tubercles or small nodules, which to the naked eye closely resemble the grey hemp-seed growths of tuberculosis ; and the disease is only recognised by a microscopic examination of the nodules obtained on *post-mortem* examination.

Diagnosis.—This must always rest upon the discovery of the streptothrix in the expectorated pus or in an abscess which points between the ribs. The presence of actinomycosis in bones, especially in those of the jaws, should arouse a strong suspicion of the nature of any pulmonary disease present at the same time.

Prognosis.—A fatal issue is to be expected, unless in those rare instances where a single localised deposit ends in an abscess which is evacuated through a bronchial tube.

Treatment.—Iodide of potassium in 20 to 30 gr. doses thrice daily has a curative influence if administered early. In rare suitable cases surgical measures, as for abscess of the lung, may be indicated.

LUNG, ASPERGILLOSIS OF

This is a rare pulmonary disease, caused by the inspiration of the spores of a fungus, *Aspergillus fumigatus*, which exists on various kinds of grain.

Aspergillosis is practically confined to the lungs, though the fungus may sometimes infect the ear and skin. It is met with in pigeon-feeders who place bird-seed of various kinds in the mouth before transferring it to the bill of the pigeon. According to Rolleston, it is also met with in hair-sorters, who employ rye-flour to dust over the hair in order to make it more easily manipulated. Occasionally millers and farm-servants working with grain suffer from the disease.

The lesions caused by the fungus resemble those found in phthisis, and the symptoms may be practically identical, the only difference in the clinical picture being the presence in the one case of the mycelium of the fungus in the sputum, and in the other of the tubercle bacillus. In a number of cases of this rare pulmonary affection both organisms have been found together, and it is probable that the explanation of this lies in the fact that the tubercular lesions had supplied a suitable nidus or culture-ground for the spores of the aspergillus, which were inhaled at a later period. This is rendered almost certain by a study of those cases of chronic pulmonary disease, as bronchiectasis and old lung-abscesses, in which the spores are found to have invaded the cavities in a secondary manner.

Baccarani recently has recorded three cases in which the disease was *primary*, no association with the tubercle bacillus existing ; and he classifies the condition, which resembled acute tuberculosis, under the heading of *Pseudo-tuberculosis*. The parasite acts as a mechanical irritant, and the fever, suppuration, and wasting are to be attributed to the inflammatory changes induced by the spores in the pulmonary tissue, and not to any toxin manufactured by them.

There are two types of the affection described : one in which the main changes are those of fibroid proliferation, as are seen in chronic interstitial pneumonia ; the other type, whilst manifesting the same lesions to a lesser extent, shows a very extensive compensatory emphysema.

The disease remains confined to the lung, though the pleura may be sometimes infected secondarily ; and the tendency is generally towards recovery after the expectoration of the fungus. But even after the removal of the fungus from the body the chronic fibroid changes induced by its irritant action may progress and cause death from injury to the lung-tissue, as in fibroid phthisis.

The treatment is that indicated in chronic tubercular disease of the lung. It is needless to say that change of environment and occupation is essential. Iodides are credited with curative properties, and some French authorities maintain that they possess the same inhibitory power over aspergillus-development as they do over the growth of the ray-fungus.

LUNG, ATROPHY OF

This condition will be found described under ' Emphysema,' of which disease it constitutes the variety known as small-lunged emphysema, which is usually but a part of the general withering met with in old age.

LUNG, CANCER OF

Carcinoma of the lung as a *primary* affection is very rare ; when it does occur, it starts usually as a cylindrical-celled carcinoma in the epithelial lining of the bronchi, or in the alveolar tissue or lymphatic glands, and involves in a diffuse manner the greater part of a lobe or of the entire lung, the opposite organ almost invariably remaining free. The endothelial type of primary carcinoma is an exceedingly rare form, developing from the lymphatic vessels of the lung.

Primary cancer may occur at an early age, following severe trauma of the thorax ; it is more frequent in men.

The growth usually first invades the lung at the root, whence it steadily spreads along the lines of the bronchi, throughout the lower lobe, only involving the apex last. The infiltrated organ becomes converted into a mass of soft or medullary cancer, not unlike white brain-tissue, in which hæmorrhagic areæ are often present.

Secondary carcinoma of the lungs is much more common, occurring in malignant disease of the breast, liver, or other organ, when it usually appears in rounded isolated masses of various sizes and of soft consistence, displacing and compressing the surrounding pulmonary tissue. In some cases the lung may become invaded by the gradual direct extension of a cancerous growth from the vertebræ, pleura, gullet, or lymphatic glands. Usually, however, the invasion takes place by means of small fragments of softening cancerous tumours being conveyed directly through the pulmonary arterioles to the lung-tissue, as evident in the form of the disease in which both lungs may be found studded throughout with nodular masses like tubercle.

The medullary masses may break down, causing irregular cavities, and often extensive hæmorrhages or gangrene of the lung. The lymphatic glands are infected, and pleuritis, with blood-stained effusion, may result. In other cases, but especially in the primary form, extensive pneumonic consolidations may surround the diseased area. A large bronchus may become blocked through pressure or by the growth directly filling up its lumen.

Symptoms. — In primary cancer the growth may cause a visible and palpable enlargement of one side of the chest in its lower zone, such as is seen in pleural effusion. There may be considerable pain, especially when the pleura is invaded ; dulness is marked, and the case may be readily mistaken for one of pleural effusion, as not unusually some exudation of fluid may be also present.

Dyspnœa and cough, with the expectoration of a jelly-like mucus, which may be often tinged with bright

blood or assume the prune-juice coloration, are often present.

The dyspnœa may become alarming and paroxysmal when the trachea or large bronchi are involved by pressure. The auscultatory signs may reveal nothing but diminished breath-sounds. The introduction of an exploring needle may show that no fluid is present, though the vocal fremitus and vocal resonance may be diminished. Sometimes, however, bronchial breathing is well marked; and where from pressure or obstruction a main bronchus becomes narrowed there may be stridor with both the inspiratory and expiratory acts.

Some enlargement of the supra-clavicular lymphatic glands, and of those in the axilla, may be observed, and not infrequently there is distension of the large superficial veins, and œdema, with lividity of the upper part of the body, or of the head, or of one upper extremity. Difficulty of swallowing from pressure on the gullet, and paralysis of the larynx from recurrent laryngeal pressure, may be observed.

In secondary cancer, symptoms and signs may be entirely absent, unless when large masses form, in which case there will be present a variable number of the above, according to the site and extent of the deposit of the neoplasm. It is only in the primary diffuse form of carcinoma that there is marked displacement of the heart observed; but even in this variety of the disease there may be retraction of the affected side simulating the physical signs met with in fibroid phthisis.

The sputum in both primary and secondary pulmonary carcinoma reveals characteristic features on microscopic examination, according to Hampeln and others, who describe numerous unpigmented rounded cells, with clear large nuclei and nucleoli, and finely granular protoplasm.

The disease runs a rapid course, death occurring usually in less than nine, and often within four, months. The usual cancerous cachexia, with anæmia, emaciation, and sometimes intermittent fever, is present.

Diagnosis.—As seen from the above list of signs and symptoms, cancer of the lung presents a varied clinical picture, which often causes great difficulty in diagnosis. In the secondary variety the discovery of a malignant growth in some other part of the body affords an obvious key to the unlocking of the anomalous and puzzling symptoms. The appearance of pulmonary cancer not rarely follows the removal of cancerous tumours from the testicle, mamma, uterus, bones, or other regions, after the expiration of a considerable interval. The history of a previous surgical operation undertaken for the removal of a growth, which was even supposed or believed to be of an innocent nature, should be carefully weighed.

Primary cancer of the lung can be differentiated from *pleurisy with effusion* only after a very careful survey of the history and study of the physical signs. The exploring needle must be brought into use, and several punctures made, before negative evidence of the presence of fluid can be accepted ; occasionally the histology of a minute fragment of tissue blocking the lumen of the needle may settle the diagnosis. Even where fluid is found in the pleura there may also be a large malignant mass at the base of the lung, and the nature of the case can only be decided, after the withdrawal of all fluid, by carefully mapping out the dulness on percussion which remains. The fluid of pleural effusion complicated or caused by cancer is often blood-stained ; but this also is seen in tubercular pleurisy, which latter effusion, however, may contain tubercle bacilli. The presence of enlargement of the lymphatic glands above the clavicle, of distension of the superficial veins, or of local œdema, characteristic cells in the sputum, and rapidly progressive marasmus, and repeated speedily occurring re-accumulations of fluid all point to cancer.

Sarcoma of the lung is much rarer than cancer, but when present in diffuse form the physical signs are practically the same. In sarcoma, a history or the presence of a sarcomatous growth elsewhere can usually be made out.

Phthisis is only likely to be mistaken for cancer affecting the apices of the lung, but in the latter the dulness, pain, and dyspnœa are usually well marked, and there may be glandular enlargement and dark-red jelly-like sputum, with cancer-cells and absence of tubercle bacilli. Cancer of the lung has been sometimes found associated with tubercle, but only in patients past middle-life.

Aneurismal tumours and growths in the mediastinum may closely simulate cancer of the lung, and the diagnosis, in the absence of pulsation, thrills, and murmurs caused by sacculations or fusiform dilatations of the main vessels, can be only arrived at after watching the slow progress of the case.

Prognosis. — This, it is needless to say, is always fatal in pulmonary carcinoma ; and treatment, save as regards amelioration of the suffering, is always futile.

LUNG, CIRRHOSIS OF.

(See under PNEUMONIA, CHRONIC.)

LUNG, COLLAPSE OF

This condition, which is also known as Atelectasis, is characterised by the absence of air in the pulmonary alveoli. It

has already been described as one of the features of broncho-pneumonia or capillary bronchitis, in which disease it is caused by obstruction of the bronchioles, the imprisoned air being afterwards absorbed. By some writers the condition is described under the title *Apneumatosis*, the term Atelectasis being reserved for the airless state of the lungs which is normal in fœtal life.

Accepting the designation 'collapse of the lung' in its usually accepted sense, it must be regarded as a *secondary* condition to obstruction of the air-passages from any condition which diminishes their lumen from within, as the presence of inflammatory products ; or from without, as the compression or occlusion produced by the pressure of tumours, pleural effusion, &c.

A similar result follows in cases of extreme debility or paralysis of the respiratory muscles ; this is seen in the atelectasis which remains after birth in weakly infants and rachitic children. Atelectasis may also be caused in robust new-born infants by the obstruction of the air-passages by meconium inspirated during the later stages of labour.

The anatomical changes have been described under 'Broncho-pneumonia,' where the scattered collapsed lobules or patches of lung-tissue may be seen depressed below the surface-level of the lung, especially in the lower lobes. Such patches are firm and airless, and sink in water ; they are of a dark-red colour, and when recent may be inflated by the blow-pipe, but when of long standing the alveolar walls become glued together, and the conditions variously described as splenisation and carnification result.

The collapse may in some instances affect an entire lobe, and in rare cases an entire lung. More or less compensatory emphysema is usually present in other portions of the pulmonary tissue. The airless condition of the lung met with in pleural and pericardial effusions, aneurismal, hydatid, ovarian, or other tumours, and in ascites which elevates the diaphragm, and in spinal curvature, is obviously described by its name—*compression* of the lung.

Symptoms.—In the new-born the chief signs are the cyanosis and diminution in the breath-sounds, with some dulness over the bases, and loss of vocal fremitus ; the dyspnœa is of the inspiratory type, the intercostal spaces, lower margin of the thorax, and infra-clavicular fossæ being retracted.

In collapse following broncho-pneumonia, pertussis, and other conditions, in addition to the signs and symptoms of the primary disorder, there may be a sudden increase in the cough, rapidity and difficulty of breathing, accompanied by some cyanosis, associated with great prostration and restlessness, as indicated by rapidity of pulse, coldness of the

extremities, a sunken condition of the eyes, and the other symptoms described upon page 152.

The physical examination of the chest will reveal the inspiratory type of the dyspnœa, with retraction of the lower margin of the thorax and depression of the intercostal spaces in inspiration. The breath-sounds may be entirely absent over the affected areæ, which are also dull on percussion. These signs and symptoms may supervene after a severe attack of coughing, and may in some cases rapidly pass away, especially in pertussis without much bronchial secretion.

The bronchial affections met with in aged patients are also liable to be complicated with pulmonary collapse, whilst in the adult and middle periods of life the phenomenon is rare, except in very debilitated subjects.

Prognosis.—This is grave in proportion to the seriousness of the primary condition, the feebleness of the patient's respiratory powers, and the extent of the area of pulmonary tissue involved.

Treatment.—In the atelectasis of the new-born infant the mouth and throat should be rapidly swabbed out in order to remove all obstructing secretions, and artificial respiration should be commenced. Douching of the chest-walls with warm and cold water alternately is a powerful agent in inducing respiration, as is also electricity when obtainable. In desperate cases insufflation of the trachea by air blown through a soft catheter may be resorted to, though the operation is not without danger. The action of vomiting tends also to pump out the obstruction in the bronchi.

In the treatment of the pulmonary collapse following broncho-pneumonia, free stimulation, emesis, cold douching, oxygen-inhalations, and the measures referred to upon page 154 should be resorted to. In aged patients, in addition to these, hypodermic injections of strychnine are indicated.

LUNG, CONGESTION OF

Hyperæmia of the lungs is either *active* or *passive*.

ACTIVE PULMONARY CONGESTION

Etiology and Pathology.—The condition is a part of most pulmonary inflammations, and is constantly met with in the early stages of pneumonia and pleuritis, and at a later period in tuberculosis and other conditions. It may follow violent muscular exercise, the inhalation of very cold or superheated air, or air charged with irritant gases ; and a deeply congested or engorged condition of the lungs is often

witnessed after death from alcoholic poisoning which has been preceded by exposure to cold.

Whether, as taught by French physicians, there exists a primary acute congestion of the lungs independent of the presence of the pneumonic micro-organisms is a matter of uncertainty. When death occurs in pneumonia before consolidation takes place the anatomical changes would suggest such an explanation of the engorgement; but a bacteriological investigation should settle the nature of the case.

The writer can recall two cases, in one of which there was no fever, where the physical signs were those met with in the first stage of pneumonia, and the symptoms suddenly set in during apparently perfect health, with great dyspnœa and cough; both lived several days without any evidence of pneumonic consolidation. A *post-mortem* examination was obtained in the non-febrile case, and no disease of the heart or vessels or of any other organ was present, the anatomical changes being strictly confined to the deeply engorged lungs. No bacteriological examinations were available at the time these cases were observed. In the primary acute pulmonary congestion described by and named after Woillez there is said to be always marked but not very high fever.

PASSIVE PULMONARY CONGESTION

This is seen in two forms: (1) the mechanical or obstructive, and (2) that due to hypostatic congestion.

Mechanical Congestion. — **Etiology and Pathology.** — In mitral lesions, as already described, the increased blood-pressure in the left auricle leads to engorgement of the pulmonary veins, which soon distends the capillaries of the lung. When the backward pressure is of long standing the alveoli become blocked by shedding of their epithelial cells; the alveolar walls and the desquamated cells are stained a deep brownish-red by altered blood-pigment. There is some increase in the interstitial fibroid tissue, causing an increased consistency and unusual toughness of the pulmonary substance. To this pathological condition the term *red induration* or splenisation of the lung is applied; at a still later stage, owing to the increasing amount of pigment present, the condition is often termed *brown induration* of the lung. Any chronic cause producing marked cardiac failure or pulmonary obstruction, as in emphysema and fibroid phthisis, will also lead to the production of this form of pulmonary induration.

With the induration is usually associated more or less pulmonary œdema, the bases of the lung suffering most.

Symptoms.—The effects of mechanical congestion usually do not become obvious till compensation breaks down, when œdema of the lung and extension of the pulmonary

engorgement will cause the dyspnœa, lividity, cough, &c., which are characteristic of the failing compensation seen in the last stages of nearly all valvular lesions. The sputum may be blood-stained, and usually will show abundance of desquamated alveolar epithelial cells, deeply coloured by altered blood-pigment.

Hypostatic congestion is often confounded with pneumonia.

Etiology and Pathology. — This condition is always secondary to some grave state of the system induced by profound debility.

The term implies that the engorgement of the lungs obeys the laws of gravity ; hence the bases are the usual site of this form of passive hyperæmia.

Some confusion exists about its pathology, since the term 'hypostatic pneumonia' is often applied to it. The main factor in its production is certainly cardiac weakness, associated with a paralytic condition of the vessels which induces 'pulmonary stasis.' The influence of gravity naturally limits the engorgement to the most dependent parts of the lung, which become of a deep purple or almost black colour. When of longer standing, inflammatory changes are liable to supervene on the state of stasis, and a low form of pneumonia sets in, which follows neither a lobar nor lobular distribution. The affected portions of the organs then become airless and may almost sink in water, exhibiting the characters recognised as *splenisation of the lung*, in which no blood oozes from its cut surface, whilst in the earlier stage the lung bleeds on section. The bronchial tubes participate in the hyperæmia, the vessels of the mucous surface being engorged, and the mucous glands are in a state of catarrhal inflammation. There is also more or less œdema present.

The microscopic changes are somewhat similar to those found in hyperæmia due to the mechanical obstruction of valvular diseases—*i.e.* there is abundant desquamated alveolar epithelium ; but this is in a state of necrosis or degeneration, so that the cell-outlines and nuclei are hardly appreciable (Aufrecht).

The subject of hypostatic congestion will be again discussed under 'Pneumonia,' and its relationship to inflammatory changes will be referred to.

The condition is very common in typhoid and other fevers, and in all affections compelling feeble aged folk to remain for a long time in bed in some unchangeable position, as is the case in extra- or intra-capsular fractures of the femur. It supervenes rapidly in cerebral hæmorrhage, spinal affections, and in alcoholic and opium poisoning where the patient's position has remained long unchanged during the comatose period, in all of which cases it may be associated with aspiration-pneumonia.

Symptoms.—The symptoms of the primary debilitating malady usually overshadow those of the pulmonary condition, but the advent of dyspnœa, cyanosis, and cough should arouse suspicion of hypostatic hyperæmia. The bases of both lungs will then be found to yield dulness on percussion; and fine moist crepitation, with sometimes feeble bronchial breathing will be audible on auscultation.

Prognosis.—This is the prognosis of the primary disease causing the malady, and it is always grave.

Treatment.—The indication in mechanical congestion is for remedies which strengthen the heart's action—*i.e.* cardiac tonics and the stimulation suitable in failing compensation. In hypostatic congestion frequent change of position is urgently demanded, in addition to cold douching or sponging of the thorax. The patient should be directed to breathe deeply, so as to cause as thorough inflation of the lungs as possible; artificial respiration may in grave acute cases be necessary.

LUNG, DUST-FIBROSIS OF

The disease of the lung produced by the inhalation of dust of various kinds will be discussed under 'Pneumonokoniosis.'

LUNG, EMBOLISM OF

This is a condition common in advanced disease involving the right side of the heart, especially where thrombosis affects the right auricle in mitral stenosis. It also occurs in thrombosis of any of the systemic veins, and more rarely from clots forming in a diseased branch of the pulmonary artery. The subject has been dealt with already in the article on 'Embolism,' on page 359, where fat and air embolism are also referred to. (See also under 'Thrombosis.')

LUNG, EMPHYSEMA OF. (See under EMPHYSEMA.)

LUNG, GANGRENE OF

Necrosis or death of a portion of the lung-tissue is to be almost always, if not invariably, regarded as secondary to some pulmonary change in the bronchi, vessels, or alveolar tissue.

Etiology and Pathology.—In considering the etiological factors capable of producing gangrene of the lung it seems highly probable that there is no solitary special agent which can be considered as operative in any case, the advent of gangrene being the resultant of several distinct forces acting at the same time.

Micro-organisms are probably always essential for the success of the gangrenous process; though these are of themselves incapable of causing necrosis unless in the presence of some marked diminution in the power of resistance of the pulmonary and bronchial tissue. Staphylococci, especially pyogenes aureus and albus, are very frequently present; streptococci and various other cocci and bacilli, also the microphyte described by Hirschler and Terray, and the fungi found by other observers, have been accredited with specific powers. Babès is probably right in his view that pulmonary gangrene can only be produced by several of these different micro-organisms operating at the same time.

The following conditions have been found so frequently to precede the advent of gangrene that they may be regarded as *predisposing* causes which prepare a way for the invasion of the microbes—viz. (1) Inflammatory processes, as those seen in croupous pneumonia and in the lower type of inflammatory action causing deglutition or aspiration pneumonia from the aspiration of septic particles of food, or secretions finding their way into the bronchi in cancer of the gullet, or perforation of the bronchi from diseased glands, or from the extension of empyema; (2) Bronchiectasis; (3) Phthisical cavities: both these latter conditions are occasionally followed by a spreading gangrene, starting in the walls of the caverns in the lung, which contain decomposing secretions; (4) Pressure of aneurismal or other tumours on the pulmonary tissue or on a main bronchus; (5) Thrombosis of the pulmonary artery, or septic emboli carried from distant parts, as in septicæmia, caries of bone, pyæmia, &c., or from pulmonary infarction; (6) Traumatism producing perforation of the thorax, or laceration of the lung-substance without external wound; (7) Gangrene of the lung may supervene upon any debilitating condition, as in protracted typhoid fever, smallpox, diabetes, and senility, especially when associated with chronic alcoholism, starvation, or exposure. It was formerly not uncommon as a sequel to injuries and diseases occurring in armies during protracted campaigns, and in prisons where pneumonia and typhus were frequent from imperfect hygienic measures and bad food.

Pathological Anatomy.—Two types of pulmonary gangrene have been recognised since the time of Laennec—*i.e.* the circumscribed and the diffuse. In the former a small portion of the pulmonary tissue becomes necrosed, turns of a dark green colour, emits a putrid odour, and at first is of firm consistence. It is bounded by a variable zone of inflamed tissue, which separates the diseased pain from the healthy lung. The green mass rapidly softens at the centre, which is transformed into a black pulp, perforation of a bronchus occurs, and the broken-down tissue is

expectorated, leaving a variably sized cavity with irregular walls, and usually containing foul-smelling green liquid secretion.

The inflamed zone becomes the seat of consolidation or induration, a false lining membrane forms, and the cavity may ultimately present the features of an ordinary pulmonary abscess or tuberculous cavern.

In the diffuse form a large portion of one lobe, or its entire bulk, becomes converted into a greenish-black, friable, œdematous mass, which merges at places imperceptibly into the healthy tissue. At several spots the dead tissue softens, and excavation occurs as the offensive, soft, pulpy, or liquid *débris* is expectorated. Often large vessels are found extending across the excavations; when they are not plugged in the original embolic process, they may be opened by necrosis of their walls, causing a fatal hæmorrhage.

Pneumonia originating at the peripheral part of the gangrenous mass may extend, involving the entire lung and leading to a fatal issue.

In both forms of gangrene of the lung the lower lobes are usually the seat of the necrotic process. With the changes already described there are also those of a more or less general inflammation of the bronchi, and there may be empyema from perforation of the pleura.

Symptoms.—The condition is preceded by the symptoms of the primary disease of the lung, whether this be tubercular, bronchiectatic, pneumonic, thrombotic, traumatic, or embolic. In the diffuse form, according to Aufrecht, there are always precedent symptoms of a non-febrile bronchial catarrh. The only reliable symptom or sign of pulmonary gangrene is the expectoration of the characteristic fetid secretion. The odour is indescribably foul, and permeates everything about the patient; it taints his breath in the intervals between his coughing, and often pollutes the air of a moderately sized room, even when the sputum is expectorated directly into a powerful disinfecting solution.

The fetid sputum, on standing, separates into three layers, the uppermost of which is thick, frothy, and opaque, and usually of a greenish-yellow colour; the middle stratum is translucent and thin or watery in consistence, and of a brownish-green hue; whilst the sedimentary layer resembles dark-coloured greenish pus. Under the microscope the sediment is seen to consist of pus-corpuscles, fat-cells, pigment-granules, fatty needle-shaped crystals mixed with yellow elastic fibres, and the numerous cocci and other micro-organisms already mentioned. Fragments of broken-down lung-tissue may be sometimes seen by the naked eye, and very occasionally firm yellowish plugs of various sizes

containing fatty needle-shaped crystals when examined under the microscope.

These characters of the sputum are not, however, to be regarded as pathognomonic of pulmonary gangrene in the absence of unmistakable shreds of pulmonary tissue, since almost identical constituents may be found in the sputum from bronchiectasis. Aufrecht lays stress upon the character of the odour, which of itself is sufficient to establish a differential diagnosis, the stench of gangrene being decidedly fæcal or intensely fetid, whilst that of putrid bronchitis resembles the odour of rotten cheese, conveying the idea of putridity.

(Of more importance, however, in the differential diagnosis are the points to be found in investigation of the history. Bronchiectasis is a chronic affection, whilst gangrene usually supervenes with more or less abruptness associated with grave prostration, and runs an acute course.)

Fever is nearly always present, sometimes reaching as high as that seen in acute pneumonia; and with it the prostration is usually well marked, especially on movement of the body.

Cough is constant and distressing, often producing grave exhaustion; decubitus is usually on the affected side, the patient lying with his shoulders well raised. Hæmoptysis occurs to a slight degree frequently during the disease, and occasionally it may be so profuse as to cause death.

Physical signs are not characteristic unless when rapid formation of cavities occurs, but the pneumonic consolidation around these may cause such dulness and alteration in the breath-sounds as may lead to mistakes in diagnosis. The signs of extensive severe bronchial inflammation are always present.

Occasionally after death localised gangrene of the lung has been found when no symptom or sign had been noticed during life, probably owing to the blocking of the bronchi in the affected spot.

Diagnosis.—The main difficulty lies in the differentiation of bronchiectasis, the chief points in which have been already detailed in the previous paragraphs.

Prognosis.—This is always serious, and in the diffuse form generally fatal. In the circumscribed form, when the site of the gangrenous cavity can be localised, surgical treatment being indicated, the outlook is decidedly good in many cases. Le Conte gives the mortality as 80 per cent. under medical and 40 per cent. under surgical treatment.

Treatment.—This, from the medical point of view, resolves itself into the disinfection of the affected area by the administration internally, and by means of sprays and inhalations, of volatile antiseptics, as indicated in bronchiectasis and abscess of the lung. Surgical measures should be employed in all cases where the gangrene is localised and where the site is within

reach. Contrary to what might be expected, operation is much more successful in cases of gangrene following pneumonia than in those which supervene upon bronchiectasis.

LUNG, HÆMORRHAGE FROM

This sign or symptom, so often met with in pulmonary diseases, aneurisms, valvular affections, hæmorrhagic infarction, and morbid states of the blood, is dealt with under 'Hæmoptysis.'

LUNG, HYDATIDS OF. (See under HYDATIDS.)

LUNG, INFLAMMATION OF.

(See under PNEUMONIA.)

LUNG, ŒDEMA OF

This condition may be associated with any form of pulmonary congestion; it is not usually present to any very marked degree in valvular disease, though in the later stages of failing compensation some amount of pulmonary œdema is always present.

Pathology.—The pathology of ordinary general œdema still requires clearing up, the proportionate influences of the nervous, blood, lymphatic, and circulatory factors being difficult of determination. In pulmonary œdema a like difficulty confronts the pathologist; and the underlying blood-condition must be an important etiological factor, since the high pressure in the vessels caused by a narrow mitral stenosis is often insufficient to produce œdema of the pulmonary tissue. Degenerate changes in the capillary-walls favouring transudation, a like condition in the lymphatic vessels retarding absorption, a diluted state of the blood-plasma, disturbed innervation of the cardiac plexus, and a failure in the muscular power of the dilated ventricle to drive the blood against the increased pressure in the pulmonary system, have all been held as operative factors.

The essential feature in the affection is the exudation of serous fluid in the air-vesicles, smaller bronchi, and in the spaces in the interstitial tissue. The affected organs are heavy and may sink in water, and pit on the pressure of the finger-tips. The base is most commonly affected, but the entire organ may be dropsical. On section, serous fluid freely oozes out. Should congestion exist, blood or blood-stained

serous fluid transudes; but the lung may be pale, and the exudate watery in appearance in some cases, especially in those of renal origin.

Etiology.—All inflammatory conditions of the lungs, as croupous and septic or aspiration pneumonia, the passive hyperæmia occurring in the last stage of valvular lesions, and the hypostatic congestion produced by debilitating diseases and unchanging postures in the aged, are complicated by more or less œdema of the pulmonary tissue. In a local or circumscribed form it is met with in the areæ of the lung surrounding tumours, infarcts, and abscess, or around regions subjected to direct pressure by aneurisms or growths outside the lung.

Bright's disease, both of the desquamative and cirrhotic types, is often associated with a rapidly fatal pulmonary œdema. Huchard states that œdema of the lung is very much more common in the cirrhotic kidney; but this is not the writer's experience, though undoubtedly it is more frequently met with in its most acute form in this type of renal disease.

The inhalation of irritant gases causes œdema, and death not very rarely follows ether anæsthesia. Pilocarpine has been often the cause of a fatal lung-œdema, but never when the precaution is taken to postpone its administration till after the skin has been made to act freely by a hot pack. Morphia, alcohol, muscarine, and iodides occasionally produce the same effect in spite of profuse skin-action.

Pulmonary œdema sometimes sets in immediately before death in apoplectic and profoundly anæmic or cachectic states, in which instances it may be regarded as part of the final agonal phenomenon.

Symptoms.—The sudden supervention of dyspnœa or orthopnœa, lividity, cough, distension of the veins in the neck, cold sweating, and the expectoration of a profuse watery or blood-stained thin serous secretion coming on in Bright's disease or any of the other primary affections before mentioned, are characteristic of an attack of acute pulmonary œdema. Where the condition is grafted on any lesion which has already produced passive congestion, the pulmonary symptoms merely become intensified and the sputum more profuse, liquid, and frothy.

There is more or less dulness over the bases of the lungs, seldom complete at any one spot, whilst in the upper thoracic region the note may be hyper-resonant. Fine and middle-sized râles are heard at first over the bases, and afterward extend upwards, the breath-sounds are diminished, and the vocal resonance feeble. At a later stage the water-logged condition of both lungs shows itself by the symptoms of impending suffocation, in which the most yielding portions of

the thorax and intercostal spaces are drawn in during inspiration, the clinical picture of the orthopnœa resembling that seen in laryngeal diphtheria.

A recurring, intermittent, or periodic type of suddenly occurring œdema of short duration, but of severe degree, is sometimes met with in disease of the aortic arch, angina pectoris, cirrhotic renal disease, and acute dilatation of the heart following severe exertion.

The diagnosis, prognosis, and treatment are those belonging to the primary disease. Sometimes death may be averted by the prompt and free incision of a large vein in the arm, or by a full dose of saturated magnesium-sulphate solution as recommended by Hay. Diaphoretics are useless, save in the form of hot mustard-packs ; diuretics must not be relied upon, but occasionally full doses of sweet spirit of nitre act beneficially by dilating the afferent vessels in the renal glomeruli. Pilocarpine should not be administered. Hypodermic medication by full doses of strychnine and digitalis is indicated when the heart shows signs of impending failure.

LUNG, PERFORATION OF

The term is employed to designate the condition in which the pulmonary pleura is placed in direct communication with the interior of the lung.

Etiology.—The condition may be the result of wounds which have also perforated the costal pleura, or it may be caused by the puncture of a broken rib, unassociated as it commonly is with any skin-laceration or incision.

Disease of the pleura, as in empyema, may lead to perforation by the extension inwards of the suppurative inflammation, and various localised conditions may lead to perforative abscesses in the same manner, as suppuration of a bronchial gland, of a tubercular vertebra, hepatic hydatids, gastric ulcer, or hepatic abscess. Aneurisms, and especially cancer of the œsophagus, may act from without in the same manner.

More commonly, however, the pleura is perforated from within by disease of the lung. In the great majority of all cases perforation of the pulmonary pleura is the secondary result of tubercular disease originating in the lung-tissue.

Pulmonary gangrene and abscesses, emphysema, cancer, infarcts, hydatids, and more rarely bronchiectasis, may all in the same manner eventuate in the formation of an opening into the pulmonary pleura. The immediate result in most cases is the entrance of air into the pleural cavity, as seen in its most typical form from the rupture of an emphysematous bleb near to the surface of the lung. Hence, perforation of the lung is practically synonymous with pneumothorax. In

the majority of cases, however, the admission of air leads to the formation of pus in the pleural cavity; the affection is then distinguished by the title *pneumopyothorax*, to differentiate it from the purulent effusion which makes its way inwards after perforation, this latter condition being known usually as *pyopneumothorax*.

Blood and serous fluid may be poured out after the admission of air from the lung to the pleura in some cases instead of a purulent exudation; hence the terms *pneumohæmothorax* and *pneumohydrothorax*.

The symptoms and signs are described under 'Pneumothorax' and 'Empyema,' where prognosis, diagnosis, and treatment are also discussed.

LUNG, SYPHILIS OF

Pathological Anatomy.—In congenital syphilis, the disease may exhibit itself in still-born infants or in those who survive birth for a few minutes or hours by the presence of the so-called white hepatisation, or pancreatisation of the lungs. The organs are bloodless, enlarged, firm, airless, and heavy, sinking rapidly in water; the alveoli are filled with epithelial cells, and the alveolar walls, as well as those of the small blood-vessels, are found greatly thickened; gummata may be also present. Aufrecht considers the alveolar changes to be the direct result of the thickening of the vascular walls.

In acquired syphilis, implication of the lung is very rare; when it does occur it is always a tertiary manifestation. Gummatous nodules resembling caseating tubercle have been found by some observers scattered throughout the lungs. In other cases changes have been observed in the pulmonary tissue, which becomes the seat of diffuse fibroid induration resembling the condition met with in chronic interstitial pneumonia, the infiltration starting at the root of the lung and progressing along the bronchi and blood-vessels and constituting the so-called syphilitic pneumonia.

Symptoms.—As a rule, syphilis of the lung is only of museum interest; most of the cases, even where the gummata are large, remain unrecognised during life. When the diffuse infiltrating form affects a large area of the lung, this is often found to be the middle lobe of the right lung, a point emphasised by Grandidier as of special diagnostic importance. The main consideration in all instances of syphilitic lung-affection must be the recognition of the disease in its tertiary manifestations in other organs, especially in the coats of the arteries.

When tertiary syphilis affects the larger bronchi, or the lower end of the trachea, it is commonly associated with a similar condition of the larynx or pharynx. The disease

probably starts from the softening of a gumma, involving the bronchial wall, and chronic changes supervene similar to those described in laryngeal syphilis, the final outcome of which is the production of bronchial stenosis by the contraction of cicatricial tissue. Sometimes necrosis of the cartilage occurs. A variable degree of peribronchial and interstitial pneumonia is always present, so that syphilis attacking the lung-tissue may become almost identical with that originating in the bronchi. To these cases, in which there is bronchial involvement without much stenosis, and considerable interstitial or peribronchial change, the term 'syphilitic phthisis' is sometimes applied. Tubercular phthisis may be also associated with chronic syphilis.

From the brief *résumé* of the pathological changes it is obvious that the signs and symptoms of pulmonary syphilis must vary within wide limits. The cough, dyspnœa, wasting, fever, sweating, hæmoptysis, and the purulent expectoration, may closely simulate phthisis, especially when there is a considerable degree of bronchiectasis present. The signs are those of consolidation of the lung, chiefly of the middle lobe, with or without evidence of bronchial dilatations.

Diagnosis.—The main point in diagnosis is the differentiation of *tubercular phthisis*. This is as a rule easy, the detection of the tubercle bacilli affording positive proof of ordinary phthisis. There are, however, difficulties in the case where the bacilli are absent, and a diagnosis of syphilis cannot with safety be made unless after repeated negative examinations conducted over a considerable period. Though the site of syphilis of the lung is often confined to the middle lobe, this cannot be always relied upon: very occasionally the apex may be involved. As already stated, tertiary syphilis may be associated with tuberculosis, and the primary laryngeal manifestation of either disease may be followed by lung-symptoms. The continued absence of bacilli in the sputum is the only method of arriving at a correct diagnosis. The effect of antisyphilitic treatment is of much value in the diagnosis in early cases.

Bronchiectasis can only be distinguished from an old syphilitic peribronchitis associated with interstitial pneumonia by a careful investigation of the history and progress of the case. Where there is evidence of stenosis of a main bronchus or of the lower portion of the trachea, accompanied by signs of dilated bronchus with putrid sputum, the syphilitic nature of the case is highly probable in the absence of evidence of aneurismal or other mediastinal tumour-pressure.

Prognosis.—This will depend largely upon the condition of other organs, being always grave where amyloid disease has supervened in the kidney and liver, or where there is evidence of bronchial or tracheal stenosis with bronchiectasis beyond

the narrowing. The outlook is good when the disease is met with in its earlier stages and subjected to vigorous treatment.

Treatment.—The indications are the same as those presenting themselves in every case of chronic syphilis. In the earlier tertiary stage mercury may be freely given for short periods where the history indicates that the metal had not been previously administered in a long continuous course during the secondary period. The main reliance, however, must be placed in all cases upon large doses of iodides given for considerable periods at a time. Even when the organic changes in the lung and bronchial tissue are such as prevent any reasonable hope of the resolution of the parts under iodide-treatment, nevertheless benefits may be expected from the action of the drug upon the accompanying bronchitis and expectoration. Where syphilis and tubercle co-exist, there cannot be any doubt about the wisdom of pushing iodide-medication.

Bronchiectasis will call for volatile antiseptics by the mouth and by inhalation, and the general constitutional treatment resolves itself into that suitable for chronic tubercular phthisis, as cod-liver oil, over-feeding, open-air living, &c. Rarely will the syphilitic cavities be within the reach of surgery.

LUNG, TUBERCULOSIS OF

This will be dealt with under the heading ‘ Phthisis.’

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